**UNITED STATES DISTRICT COURT**
**SOUTHERN DISTRICT OF OHIO**
**WESTERN DIVISION**

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| **MELANIE BECKEMEYER**, | ][ | CASE NO. 1:17-cv-00695 |
| Plaintiff, | ][ | JUDGE BARRETT |
| v. | ][ | |
| **GELCO CORPORATION,** *etc.*, | ][ | **AFFIDAVIT OF ANDREW SAXON, M.D.** |
| Defendant. | ][ | |

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I, Andrew Saxon, M.D., having been duly sworn do here hereby swear, affirm, depose and state as follows:

**1. Background education, experience, and training relevant to expertise in this case.**

I am a licensed physician in the State of California specializing in the area of Clinical Immunology & Allergy. I am the founder and emeritus chief of the Division of Clinical Immunology and Allergy at the UCLA School of Medicine, Los Angeles, California. I am currently a Professor of Medicine at the David Geffen School of Medicine at UCLA.

I received my medical degree from Harvard Medical School in Boston, Massachusetts, in 1972. I completed my residency in internal medicine at Harbor General Hospital in Los Angeles, California, in 1975, and my post-doctoral training in immunology in the Department of Microbiology and Immunology at the UCLA School of Medicine in 1977. I am a licensed physician in California through the National Board of Medical Examiners. I am board certified in (1) Internal Medicine by the American Board of Internal Medicine, (2) Allergy and Immunology by the American Board of Allergy and Immunology, and (3) Diagnostic Laboratory Immunology by the American Board of Diagnostic Laboratory Immunology. I founded the Division of Clinical Immunology and Allergy in the Department of Medicine at UCLA in 1977 and served

as its Chairman for 30 years. I also founded the UCLA Asthma, Allergy, and Immunologic Disease Center and served as Director until the time of my retirement. I have published over 190 scientific papers in peer-reviewed journals in the field of immunology and I provide many editorial/review services for such journals including being Editor in Chief of Clinical Immunology, the official journal of the Clinical Immunology Society for over a decade. I also have served on peer-review funding committees for the National Institutes of Health and other organizations as well as chair the Allergy/Asthma section of the Immune Tolerance Network, a major undertaking by the National Institutes of Health and private institutions to discover true cures for immune mediated disorders.

I am a co-author of the American College of Occupational & Environment Medicine (ACOEM) original Position Statement entitled: Adverse Human Health Effects Associated with Molds in the Indoor Environment that appeared in 2002. ACOEM represents more than 6,000 physicians and other health care professionals and is the nation's largest medical society of individuals specializing in the field of occupational and environmental medicine. I am also a co-author of the American Academy of Allergy Asthma and Immunology's (AAAAI) official Position statement entitled "The Health Effects of Molds" which appeared in 2006. The AAAAI, with over 5000 members, is the nation's largest medical subspecialty society specifically dealing with the allergic and immune aspects of mold exposures.

Further elaboration of my professional background, prior publications and credentials is given in the attached true and correct copy of my curriculum vitae and bibliography (**Exhibit "1"**).

**2. Data/material reviewed and relied upon in forming opinions.**

A. The basis of my testimony includes my education, clinical and basic science training, experience, and review of both basic and clinical studies regarding humans and the immune system. This includes basic studies performed in the test tube and in animals regarding mold and related substances, my own research in human and animal immune reactivity, review of the exhibits, depositions and opinions of identified plaintiff experts, my extensive reading in the areas of immunology which includes allergy, autoimmunity, cancer of the immune system (lymphomas), and related areas, and my clinical experience

including the diagnosis and management of patients with immunologically related disorders.

B. Included in this is extensive analysis of the literature relating to mold/fungal related illness in humans including the Institute of Medicine's 2004 Publication "Damp indoor spaces and Health", the American College of Medical Toxicology's Statement in Support of the Institute of Medicine's report and the 2009 World Health Organization (2009) "Guidelines for Indoor Air Quality: Dampness and Mould." I am also relying upon the 2017 Association of Scientific Medical Societies guideline, "Medical diagnostics for indoor mold exposure, the Position Statement of the American College of Occupational and Environmental Medicine promulgated by the Society in 2011 and the 2006 Position Statement of the American Academy of Allergy, Asthma and Immunology, Asthma and Immunology

C. I am also relying upon my analysis of the case-specific materials provided to me. This includes:

C1. Medical records of the plaintiff Melanie Ann Beckemeyer PharmD from the following sources that span the timeframe March 2001 through July 2018.

- Allergy and Asthma Specialty Group.
- Bernstein Allergy Group.
- Bethesda Alcohol and Drug Treatment (no records)
- Huber personalized medicine (Dr. Gray Huber, Integrative Medicine)
- Kroger Little Clinic.
- Liberty Urgent Care.
- Dr. Harold T. Pretorius.
- Plaintiff produced 2315 pages of medical documents that include the records of Dr. Craig P. Cleveland.
- Blue Ash Family Medicine
- Report of Richard Sexton, PhD of April 2017.

C2. Environmental records

- The Report of Jeremy Porter, PMP dated 11.6.18
- Report from Ecostratum dated 7.25.18 for vehicle testing on 7.9.18

C3. Legal Documents

- Complaint filed in this matter.
- Discovery responses filed in this matter
- Depositions of Ms. Melanie Beckemeyer, PharmD. dated 9.27.18 and 11.5.18
- Expert Report of Dr. Scott McMahon in Beckemeyer vs. GELCO 8.31.18
- Expert Report of Dr. Scott McMahon in Fleming v Baker 3.29.17
- Deposition Testimony of Dr. Scott McMahon in Fleming v Baker 4.7.17
- Depositions of Scott W. McMahon in Courcelle v. CW NOLA PROPERTIES dated 8.18.17 and 5.18.18
- Declaration Of Scott W. Mc Mahon, MD in Courcelle v. CW NOLA Properties Dated 8.15.17
- United States District Court Western District of Washington At Seattle, Court order 16356136 dated 8.13.15 in Haneet Kumar, et al., v. Williams Portfolio 7, Inc.

3. **The basis for my opinions.**

The basis of my opinions in this in this case includes my; education, training in basic sciences and medicine, experience in general as board-certified specialist in internal medicine, allergy and immunology, and diagnostic laboratory immunology and as specifically related to mold and mycotoxin exposure, review. It also include my analysis of published peer reviewed and accepted literature on the effects of molds and mycotoxins on a broad range of mammalian species including humans, general knowledge of the adverse effects of chemicals, bacteria, molds, and toxins on mammalian species including humans, and records reviewed in this case as outlined in item #2 above.

4. **Overview of mold**.

Mold and mold spores (reproductive parts released by molds) are ubiquitous in the air and soil in all environments and settings. Molds fill every ecological niche on earth with the types and levels of specific molds varying with climatic conditions. Molds grow best in warm, damp, and humid conditions, and spread and reproduce by making spores. Molds serve a critical role in the

recycling of organic materials without which life on this planet could not be sustained. Estimates of the number of mold species range from tens of thousands to a hundred thousand or more. Mycotoxins are small non-volatile (i.e. not gaseous) chemicals that are byproducts produced by a limited number of mold species and are only produced by those molds under certain conditions, i.e. they are not constitutively produced. For example, of the over 500 species in the Genus *Aspergillus*, less than 20 produce its major mycotoxin, aflatoxin. Various mycotoxins in low levels are present throughout the food chain due to mold growing on crops.

5. **Mechanism of adverse effects from mold and mold byproducts**.

Adverse health effects from mold and their byproducts occur via three main mechanisms: Immune, Infectious and Irritant/Toxic. Each of these can be addressed in terms of whether Ms. Beckemeyer had/has evidence for an adverse health effect from mold.

A. Immune effects: There are three immune mediated diseases related to molds. First is typical inhalant "allergy" that causes allergic rhinitis and allergic asthma and occurs in genetically predisposed persons who make up about 40% of the population. These allergic airway symptoms are due to the result of the production of IgE (allergic) antibodies to ubiquitous aeroallergens such as pollens, animal dander, dust mites or mold spores. Allergy to mold occurs in about 10% of the population. The other two forms of immune adverse health effect from exposure to mold or mold byproducts are rare conditions known as allergic bronchopulmonary aspergillosis (ABPA) and hypersensitivity pneumonitis in which there is an overly strong immune response in the lung to the harmless mold spores that lead to a pneumonia -like picture.

   Whether Ms. Beckemeyer has an allergy to mold among other allergies to inhalants is unclear from the data in her records (discussed below). Even if she does, such an allergy would not account the vast spectrum of her symptoms nor their persistence. There is no indication of Ms. Beckemeyer having ABPA or hypersensitivity pneumonitis.

B. Infection: Molds commonly cause superficial infections such as toenail or skin infections (ringworm). Four unusual molds in specific outdoor geographic regions cause serious infections – primarily pneumonias. However, in profoundly immunocompromised

persons, e.g. those on chemotherapy or following bone marrow transplantation, a host of common molds can cause serious infections. None of these mold infection issues relate to one's indoor residence or vehicle.

Ms. Beckemeyer has had no mold infections. In fact, the records do not even reflect that she has had problems with common superficial mold infections such as toenail or skin infections. I would add that any oral or vaginal candida infections she may have had were due to a yeast, Candida albicans that is not a mold. That yeast is resident on normal mucosal surfaces in humans but overgrows to become an infection under a variety of circumstances unrelated to ambient environmental conditions.

C. i. Irritant effects:

Irritant effects from mold can result from inhalation of a very large concentration of mold particulates in the air wherein the spores essentially become nuisance dust. Levels in the millions of spores/$m^3$ or more would be required for this effect, levels far beyond anything found in the vehicle in question. Additionally, in high enough concentrations, mold derived volatile organic compounds (VOCs), e.g. gasses that smell and give rise to the musty odor associated with mold, can cause local irritant effects, usually of the wet membranes of the eye, nose and mouth. However, VOC irritant effects generally occur at levels hundreds to thousands of times higher than the odor threshold. Furthermore, even if a person had had some local irritant effects from VOCs, albeit there is no data to support that supposition, such effects would have been transient by definition, and would have disappeared within a week or two of Ms. Beckemeyer no longer being in the vehicle in question.

ii. Toxic effects: Toxic effects from inhalation of airborne mold spores in a residential setting or a motor vehicle essentially does not occur. This is simply a result of dose considerations; i.e. i.) the number of spores a person can inhale, ii.) the toxicity of mycotoxins on molar basis, iii.) the maximum amount of mycotoxin per spore (Kelman BJ, et al. Int J Toxicol. 23:3-10, 2004,), iv.) the half-life of mycotoxins in humans, and v.) the no observed effect level (NOEL) for even the most potent mycotoxins (Hardin BD, et al. J. Toxicol. Environ. Health A. 72:585-98, 2009. The calculations from these

studies suggests that even under the worst extreme scenario (all mold spores making the most potent known mycotoxin, inhalation of maximum amount 24/7, all spores breathed deposited and 100% of toxin absorbed) for an adult, it would take between 30,000 to >1,000,000 spores/m3 of these spores to have an adverse health effect. Compare that to the level of spores found by the Plaintiff's CIH in the vehicle air in 2018 - the airborne/respirable levels had a total 1300 spores/m3 with the majority were harmless mushroom spores, i.e. basidiospores. Of the mold spores found in the air, only those of Chaetomium (27 spores/m3) are associated with mycotoxin production. Thus the level of potential mycotoxin producing spores in the vehicle when tested was thousands of times below that at which any toxin related adverse health effect might occur.

As opposed in inhalation, ingestion of mycotoxins in food is a daily occurrence as various mycotoxin-producing molds grows on foodstuffs, e.g. peanuts, cereals, cheese etc., during their production. Indeed, the FDA and European Union has established limits for certain mycotoxin levels in foods. The level of mycotoxins in human food is generally quite low and of no health concern. However, there are occasionally outbreaks of mycotoxin poisoning in populations when starving persons have little option but to eat highly spoiled food.

Given the factors discussed above in this section, it is not tenable to propose more likely than not that irritancy or toxicity resulting from inhalation of mold spores or their byproducts in the air from the vehicle in question as causing Ms. Beckemeyer's symptomatology.

Overall, the generally accepted mechanisms for mold and/or mold byproduct induced adverse health effects are not quantitatively or qualitatively relevant to Ms. Beckemeyer's primary medical issues, i.e. her complaints do not even potentially relate to the generally accepted mechanisms of mold induced adverse health effects other than possibly some mild upper airway allergy.

6. **Medical issues that Ms. Berkemeyer had/has as related to her complaints in this matter.**

Hypertension - high blood pressure with secondary cognitive issues secondary to small vessel

ischemic changes. Ms. Beckemeyer has had high blood pressure since at least 2001 and has been maintained on long-term anti-hypertensive medication (Irbesartan most recently). Common side effects of Irbesartan include dizziness, fatigue, indigestion, diarrhea and heartburn. An MRI of her brain on 6.13.17 showed "*microangiopathic white matter disease consistent with chronic small vessel ischemia, most often seen in manifestation of hypertensive atherosclerosis or metabolic factors*." There was also mild right greater than left hippocampal atrophy that potentially may correlate with memory symptoms possibly related to early manifestation of mild cognitive impairment syndrome.

Ms. Beckemeyer has long-standing hypertension with underlying microvascular brain changes resulting in a mild impairment of certain cognitive functions, a very common scenario in a 63-year-old person. Another factor driving in Ms. Beckemeyer's cognitive complaints is her response to anxiety/stress as discussed below.

Anxiety, Panic Attacks and Depression with Somatic Symptom Disorder. Ms. Beckemeyer records show that she has had problems with depression, anxiety and panic attacks since September of 2011, long before the alleged exposure in this case. At that time, she reported she felt that "she cannot complete tasks that require organization and higher levels of cognition in a reasonable amount of time due to ongoing anxiety." She was begun on an anti-depressant Celexa and was on it through 7.1.16. By mid-2017, Ms. Beckemeyer had developed a whole panoply of ongoing symptoms that she felt were related to her exposure to the vehicle in question which had ended in September 2016. The symptoms she reported included disturbed sleep, fatigue, joint pain/swelling, bladder problems, buzzing/ringing/ear pain, blurry vision, disorientation, forgetfulness, loss of libido, unexplained hair loss, vertigo, confusion, difficulty with concentration, and swollen glands. She endorsed mood swings/irritability/depression at that time as well.

Ms. Beckemeyer has a generalized anxiety disorder with panic attacks and major depressive illness. Notably, when anxious in the past prior to the alleged exposure, she reported cognitive symptoms very similar to what she reports now. Her long standing anxiety/mood issues color and magnify her symptoms, e.g. fatigue from medications, cognitive changes etc. as well drive

such symptoms on their own as such mood issues do in all patients. Ms. Beckemeyer appears to have developed a somatic symptom disorder focused on her belief about the injury she received from her mold exposure in the vehicle in question.

Treatment with thyroid hormone. Ms. Beckemeyer has been on thyroid hormone replacement medication for many years for unclear reasons. Ms. Beckemeyer's medical records also show that in 2017 she was taking too much thyroid hormone as documented by her very low TSH.

If Ms. Beckemeyer actually does have underlying thyroid disease, which is not evident, it was pre-existing and any thyroid issues she may have are not related to exposure to mold, mold byproducts or ambient bacteria. Overtreatment with thyroid hormone as documented in 2017 will have made her hypertension and her anxiety worse.

Vertigo/disequilibrium/dizziness. Ms. Beckemeyer reports she first had problems with vertigo and disequilibrium when she was diagnosed with Meniere's disease in her 30s. On 9.18.15, Ms. Beckemeyer saw her primary care physician complaining of ear pressure and being off balance that she described as feeling "swimmy." She was treated for presumed eustachian tube dysfunction. On 9.29.15, Ms. Beckemeyer was seen in urgent care complaining of problems with wooziness and vertigo. All this was before the alleged exposure. Then in September of 2016, she complained of increasing light-headedness and dizziness. Over the ensuing months, she had an extensive work up for this including a neuro-otology consultation and no specific etiology was found for her complaints of light-headedness and dizziness.

Ms. Beckemeyer's more recent complaints of feeling dizzy and lightheaded are most likely a manifestation of both her microvascular CNS disease that is secondary to her long standing hypertension and her long standing anxiety/stress.

Upper Respiratory and external ocular symptoms: Ms. Beckemeyer saw an allergist in 2001and reported that she had had "allergies" for more than 10 years, clearly long predating her alleged mold exposure. Ms. Beckemeyer had allergy testing in 2004 but the test records are incomplete and uninterpretable. Ms. Beckemeyer had epicutaneous allergy testing on 12.27.16 with only the molds Epicoccum and Fusarium recorded as "positive" at 3+ but there is no reading scale for these tests nor was there a record of the necessary positive (histamine) and negative (saline)

control results, which makes them open to question.

The accessible data are uninterpretable as to whether Ms. Beckemeyer is an allergic (atopic) person with allergic rhinitis and allergic conjunctivitis given the problematic test results. Ms. Beckemeyer does not have asthma, allergic or otherwise. I would not rely upon the available allergy test results to make or exclude a diagnosis of inhalant allergy. This could be resolved by Ms. Beckemeyer undergoing reliable allergy blood testing done with the ImmunoCap II or equivalent third generation allergy blood testing. Even if Ms. Beckemeyer is an allergic subject and the epicutaneous testing is presumed to be accurate, the only allergens identified by this testing were Epicoccum and Fusarium. These were not found in the air in the vehicle and any exposure to these in the vehicle in question would have been far less than her exposure from air outside her vehicle as discussed in Mr. Porter's report.

7. **Use of Differential Diagnosis to establishing general and specific causation and "Chronic Immune Response Syndrome" or "CIRS."**

A. Differential Diagnosis: Differential diagnosis is the medical parallel of the scientific method in which one makes a hypothesis, tests that hypothesis experimentally and then rejects or accepts the hypothesis. The "differential diagnosis" is a list of likely generally accepted medical conditions, given the evidence in hand, with the working diagnosis (hypothesis as to what is wrong with the patient) being the most likely choice based on the currently available data. The physician then gathers valid scientific data (additional history, physical findings and/or laboratory tests to confirm or exclude that leading diagnosis.

On page 18 of his report, Dr. McMahon alleges he applies "differential diagnosis" to arrive at his final diagnostic impression of Ms. Beckemeyer having "Chronic Immune Response Syndrome" or "CIRS" as the overarching cause of her multitude of complaints. However, the record in this matter clearly demonstrates that Dr. McMahon is not using differential diagnosis in a valid or acceptable fashion. Thus while he claims to exclude other appropriate causes before reaching his conclusion, this is not the case. A cogent example of Dr. McMahon's failure to use differential diagnosis appropriately is Dr. McMahon's dismissal of hypertension as playing a role in Ms. Beckemeyer's symptoms. However, her testing shows changes indicative of microangiopathic changes in the brain that are common in hypertension

and that is generally accepted to cause in mild cognitive impairment. Instead, Dr. McMahon ascribes her neurocognitive issues to "CIRS" Another cogent example is Dr. McMahon's failure to appropriately consider Ms. Beckemeyer's pre-existing and long standing generalized anxiety disorder and depression in the genesis of her multiple somatic complaints. Generalized anxiety disorder and depression are the two most common forms of psychiatric illness in our populations. These disorders commonly give rise to multiple somatic symptoms as had been reported by Ms. Beckemeyer five years before her alleged exposure. Dr. McMahon again simply ascribes her multiple somatic symptoms to "CIRS", a concept that is not-generally accepted.

Thus Dr. McMahon, while ignoring/dismissing appropriate diagnoses, focuses with laser-like certainly on a single all-encompassing not-generally accepted pseudo-diagnosis of CIRS. In doing so, Dr. McMahon is not performing differential diagnosis as it is generally accepted in medicine. By not following the accepted procedure of differential diagnosis, Dr. McMahon comes to conclusions that cannot be relied upon in making his diagnosis of so-called "CIRS".

Dr. McMahon says that there is no other **single entity** other than CIRS that could be responsible for Ms. Beckemeyer's entire myriad of complaints. This proposition is based on a false premise; one need not have a single true pathophysiologic disease that encompasses all of a patient's medical issues. Indeed, as discussed earlier in Section 6, Ms. Beckemeyer has a number of distinct medical issues, each of which needs to be addressed individually. Such a set of different disease processes is typical for 60-year-old individuals.

B. CIRS is not a generally accepted medical condition. The lack of validity of the CIRS as a valid medical entity is discussed at length in section IIID of my report (Alternative/fringe health care and pseudo-diagnosis of Chronic Inflammatory Response Syndrome, aka "CIRS", Exhibit 3). Briefly, CIRS was invented by a former practicing family physician, Dr. Richie Shoemaker. Much of Dr. McMahon report is simply a recitation of the ideas put forth by Dr. Shoemaker who for years has been marketing himself and his "patent pending" diagnosis of CIRS (United States Patent Application Publication, Pub. No.: US 2014/0046143 A1 Shoemaker et al., Pub. Date: Feb. 13, 2014, Methods For Diagnosing, Treating, And Monitoring Chronic Inflammatory Response Syndrome). This alleged condition that Dr.

McMahon currently calls Chronic Immune Response Syndrome or CIRS is not a generally accepted entity in the medical community. CIRS is a diagnosis that is unique to Dr. Shoemaker and his followers such as Dr. McMahon.

As proposed by Dr. Shoemaker and regurgitated by Dr. McMahon, neither of whom has had any formal post graduate education or training in immunology, clinical immunology or internal medicine, CIRS is an sustained uncontrolled activation of the innate immune system due to indoor and only indoor exposure to any of a host of potential microbes/microbial products. Additionally, they propose that exposure to such microbes/microbial products in the future will lead to enhanced exacerbations of subjects' CIRS. The claim that CIRS is a self- sustaining reaction of the innate immune system is a fatally flawed idea that is in direct conflict with what is known about the human innate immune response. The battery of tests Dr. McMahon uses to support his diagnosis of CIRS are not generally accepted methodology for any purpose in clinical medicine much less for the purpose for which he employs them. Dr McMahon's claim there is no detectable lower limit for an exposure that can induce CIRS and that immune responses in CIRS and in immunology in general fail to follow dose response relationships is profoundly mistaken and in direct contrast what is known. All medicine including toxicology and immunology involve a dose response. Additionally, the claim by Dr. McMahon and other CIRS proponents that CIRS only occurs with indoor exposure is completely illogical. The literature that Dr. McMahon cites as actually supporting his opinions regarding CIRS has been generated by Drs. Mahan, Shoemaker and their colleagues while true experts in the relevant fields do not generally accept their work as reliable. Dr. McMahon's belief that CIRS can explain everything that troubles Ms. Beckemeyer (or anyone else) is based on the fact the hypothetical construct of CIRS that has no boundaries. As a result, the rubric of CIRS, such as it is, can be applied to any person to explain all alleged issues, i.e. is all-encompassing, and becomes meaningless it does not represent a separately definable disease process.

Thus, we have Dr. Scott McMahon, a pediatrician in Roswell, New Mexico, reporting that he has seen hundreds of patients who he has diagnosed with CIRS, an alleged immune mediated disorder from indoor (and only "indoor") exposure to mold/dampness. Yet no certified

clinical immunologist has ever vetted this diagnosis. So is there really an epidemic of CIRS in Roswell, New Mexico? Or is Dr. McMahon, by using "differential diagnosis" in a way that is generally not accepted, the cause of this "CIRS epidemic?"

8. **General causation**

As discussed earlier in Section 5, mold and/or mold byproducts and dampness related organisms can cause a limited known adverse health outcomes/medical issues. However, those generally accepted medical effects are not the primary health effects at issue here. At issue in this matter is whether mold/mold byproducts and/or dampness related organisms cause 1) non-infectious neurological conditions including cognitive impairment, 2) generalized somatic symptoms, e.g. fatigue, and/or 3) dizziness/vertigo. The reliable medical literature clearly does not support such conclusions. This is nicely summarized in the analyses of the Institute of Medicine (IOM), "Damp indoor spaces and Health" (Exec summary Table ES-1 and ES2, **Exhibit "2"**), the World Health Organization "Guidelines for Indoor Air Quality: Dampness and Mould." and the Association of Scientific Medical Societies Guideline publication. None of those authoritative documents found even an association between mold or dampness exposure and neurological or generalized somatic symptoms (or a host of other health outcomes.) Indeed the IOM found "*Inadequate or Insufficient Evidence to Determine Whether an Association Exists*" for these endpoints. This is in contrast to respiratory symptoms where there was "*Sufficient Evidence of an Association*" though the IOM did not feel the data reached the level of "*Sufficient Evidence of a Causal Relationship.*"

9. **Specific causation**

Given that there is no basis for general causation for mold, mold byproducts and/or dampness related organisms with non-infectious adverse health outcomes except an association for respiratory outcomes, specific causation of Ms. Beckemeyer's non-respiratory issues is moot. Furthermore, there are well-accepted causal relationships between her known medical conditions (discussed in Section 6) and her complaints. In terms of Ms. Beckemeyer's respiratory complaints and her alleged exposure in 2016, specific causation cannot be established to a reasonable degree of medical probability given the lack of valid data in that regard both in terms of the diagnosis of allergy and the potential exposures as discussed in Section 6 and Section 10. To propose that Ms. Beckemeyer had respiratory symptoms based on alleged exposures in 2016 would be purely

speculative. Furthermore, even if Ms. Beckemeyer were to have had some increased respiratory symptoms related to 2016 alleged exposure, those symptoms would have been resolved within weeks of terminating the alleged exposure.

10. **Exposure data**

The relevant metric for potential human exposure to mold/mold byproducts and subsequent dose calculations is sampling of respirable air. While testing for molds and their byproducts on surfaces in a vehicle or home or inside walls may have relevance for remediation efforts, there is no way to determine or even estimate the levels of human exposure from such sources and to attempt to do so is speculation and not generally accepted methodology for assessing human exposure. Similarly, the respirable air sampling must be in a relevant timeframe in terms of when the individual was in the location sampled. One cannot sample locations months or years after the individual was there and say what was present when the subject was there.

There are no data regarding mold or other microbial materials in vehicle in question during Ms. Beckemeyer's period of use in 2016 much less any data that the amounts were at a level sufficient to cause an adverse human health effect. It is impossible to try to derive a valid opinion as to Ms. Beckemeyer's vehicular exposure based on data obtained twenty-two months or later after her last known use. Yet this is what Dr. McMahon has inappropriately done. He has no scientific basis for opining about the mold levels in the vehicle while Plaintiff used it in 2016. Dr. McMahon forms an invalid opinion based on data from 2018 testing data and the Plaintiff's subjective report of symptoms during her use in 2016. Defining exposure based on symptoms as Dr. McMahon has done is known as "backwards toxicology", a classic error made by those with no real expert knowledge of toxicology. Dr. McMahon's attempt to "reverse extrapolate" the presence of mold and/or mold levels in the vehicle is fatally flawed as it is not based on accepted methodology for determining specific causation and as a result his opinions in this regard are invalid and cannot be relied upon.

11. **Conclusion**

It is my opinion to a reasonable degree of medical probability, based upon the records and documents available to me, that there is no valid objective evidence to support Plaintiff's allegation

that her complaints were or are adverse health effects related to her exposure to mold, mold byproducts or other microbial products in the vehicle she used in 2016.

It is my opinion to a reasonable degree of medical probability, based upon the records and documents available to me, that there is no valid objective evidence to support Plaintiff's allegation of complaints due to mold related adverse health effects. It is more likely than not that the Plaintiffs' complaints were not caused by exposure to mold, mold byproducts or other microbial products.

12. **Exhibits**

- Attached hereto as **Exhibit 1** is an authentic duplicate of my most recent Curriculum Vitae.
- Attached hereto as **Exhibit 2** is an authentic duplicate of Executive Summary of the Institute of Medicine report "Damp indoor spaces and Health"
- Attached hereto as **Exhibit 3** is an authentic duplicate of my November 22, 2018 report in this matter, which further expounds on my opinions and the bases for them.

**FURTHER AFFIANT SAYETH NAUGHT.**

ANDREW SAXON, M.D.

**SUBSCRIBED AND SWORN TO before this, the 15 day of April 2019.**

SUZETTE SOTO
Notary Public – California
Los Angeles County
Commission # 2184823
My Comm. Expires Feb 26, 2021

**NOTARY PUBLIC**

# Exhibit 1

# Andrew Saxon, M.D.

Professor of Medicine
Chief Emeritus
Clinical Immunology/Allergy
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UCLA School of Medicine
52-221 Center for the Health Sciences
Los Angeles, CA 90095-1690
Tel: (310) 367-9736 Fax: (310) 206-8622
Email: asaxon@mednet.ucla.edu

President, Secretary, & Founder
Sixal Incorporated

Board of Directors, PADI Foundation

Advisory Board, Summit Street Medical

Scientific Advisory Board, Stellar Biotech.

## EDUCATION:

- Graduate, Deerfield Academy, Deerfield, Massachusetts, 1964
  - Cum Laude Society, 1963, 1964
  - Bausch and Lomb Science Award, 1964
- A.B., Anthropology, Dartmouth College, Hanover, New Hampshire, 1968
  - Phi Beta Kappa, 1968
  - A.B. Magna Cum Laude
  - Distinction in Anthropology
  - Founding Fathers Scholar and Scholarship
- Archaeology/Anthropology (First Class Honors), University of Otago
  - Dunedin, New Zealand, 1967
- B.M.S., Dartmouth Medical School, Hanover, New Hampshire, 1970
  - Dean's Medal as First in Class
- M.D., Harvard Medical School, Boston, Massachusetts, 1972
  - Boylston Society, 1971 - 1972

## POSTGRADUATE TRAINING:

- Intern, Straight Medicine, Harbor General Hospital
  - Los Angeles, California, 1972 - 1973
  - Outstanding Medical Intern Award
- Resident, Internal Medicine, Harbor General Hospital
  - Los Angeles, California, 1973 - 1975
- Postdoctoral Scholar in Immunology
  - Department of Microbiology and Immunology
  - UCLA School of Medicine
  - Los Angeles, California, 1975 - 1977

## CERTIFICATION AND LICENSING:

- National Board of Medical Examiners (#126793), June 1973
- Licentiate (#G24948), State of California, July 1973
- Diplomate American Board of Internal Medicine (#49823), June 1975
- Diplomate American Board of Allergy and Immunology (#1967), October 1979
- Diplomate American Board of Diagnostic Laboratory Immunology (#0134), October 1990

## PUBLICATIONS

- Approximately 190 peer-reviewed research publications

## PATENTS

- Approximately 10 related to development of agents for the manipulation of the human immune response. Examples follow:
  - "Fusion Molecules and Methods for Treatment of Immune Disease" •US patent 7,265,208, 7,534,440 Issued April 19, 2009.
  - "Modified Fusion Molecules for Treatment of Allergic Disease". US application 11/050,113, PCT application 06/03562

## PROFESSIONAL APPOINTMENTS:

- Professor of Medicine Emeritus (rehire), Department of Medicine, UCLA School of Medicine, 2006.
- Executive Director, UCLA Food and Drug Allergy Care Center, 2010 - 2016
- Chief Scientific Officer, Tunitas Therapeutics, 2008 - present
- Scientific Advisory Board, Stellar Biotechnology, 2010 – present, Chairman 2010- 2014
- Professor of Medicine, Department of Medicine, UCLA School of Medicine, 1984 – present.
- Chief, Division of Clinical Immunology/Allergy, UCLA School of Medicine, 1978 – 2006.
- Associate, UCLA Molecular Biology Institute, 1985 – 2013.
- Member, UCLA Jonsson Comprehensive Cancer Center, 1983 – 2006, Associate 2106- present.
- Associate Professor of Medicine, Department of Medicine, UCLA School of Medicine, 1980 – 1984.
- Acting Chief, Division of Clinical Immunology-Allergy, UCLA School of Medicine, 1977 - 1978
- Assistant Professor of Medicine, Department of Medicine, UCLA School of Medicine, 1977 – 1980
- Chief Scientific Officer and Founder, Tunitas Therapeutics, 2008 – 2016
- President and Founder, Sixal Incorporated, 2011 - present

## PROFESSIONAL ACTIVITIES, UCLA:

### Division of Clinical Immunology/Allergy Activities:

- Founder: Division of Clinical Immunology-Allergy, Department of Medicine, UCLA School of Medicine, 1977. The activity included 6 full-time faculty members and a broad array of research scientists, technicians, and administrative support persons. I have been funded continuously by the NIH since 1977 and currently have been awarded 2 NIH research grants

- Established the post-doctoral training program in Adult Clinical Immunology and Allergy. This was later combined with the ongoing Pediatric training program so that now I was Co-Director of the Conjoint Adult/Pediatric training program in Allergy and Clinical Immunology for about 25 years. Initiated the UCLA Basic and Clinical Immunology conjoint teaching in 1977 that is now carried out in conjunction with the Department of Pediatrics.

**Departmental Activities (selected):**

- Department of Medicine Division Chiefs Committee, 1977 - 2006
- Department of Medicine Subspecialties Committee, 1999 - 2006
- Department of Medicine Honors Committee 1984 - 1987
- Department of Medicine Curriculum Committee 1989 - 1991
- Department of Medicine Chairman Search Committee 1991 - 1992

**School of Medicine and Campus Activities (selected):**

- Executive Board, UCLA Academic Senate, 1983 - 1984
- Interim Director - UCLA AIDS Center, 1985
- Associate Director - UCLA AIDS Center, 1986 - 1990
- Howard Hughes Faculty Search Committee, 1986 - 1991
- Dean's Bridge Grant Selection Committee 1995- 2010
- Co-Director, Center for Interdisciplinary Research in Immunologic Diseases Grant (CIRID) at UCLA, involving investigators from multiple School of Medicine Departments, as well as the School of Nursing, 1988 - to 1993.
- Director, UCLA Asthma, Allergy and Immunologic Disease Center 1993 – 2006, Co Director 2006 - present. This Center, funded by the National Institutes of Health (National Institute of Allergy and Infectious Disease and the National Institute Environmental Health and Sciences) is dedicated to determining the relationship between environmental factors, the immune system and specific disease processes
- Principal Investigator: Basic and Clinical Immunology Training Grant, providing for graduate and post-graduate training positions to training preceptors in both the School of Medicine and the College of Letters and Science, 1990 - 2006
- Numerous search committees.

## PROFESSIONAL ACTIVITIES, OUTSIDE UCLA:

**National Scholarly Organization Activities (selected):**

- American Academy of Allergy and Immunology, Basic and Clinical Immunology Section, Vice-Chair 1989 - 1990; Chair 1990 - 1991
- American Academy of Allergy and Immunology, Committees (active) Research Awards committee - Chair, Nominating committee, Primary Immunodeficiency Disease committee, Clinical and Diagnostic Immunology committee.
- American Association of Immunologists, Program Committee - Clinical Immunology Section, Co-Chair 1994 - 1996
- American Association of Immunologists, Program Committee - Immediate Hypersensitivity Section, Co-Chair 1990 - 1991; Chair 1991 - 1993
- American Federation for Clinical Research, National Counselor, 1981 - 1986
- American Federation for Clinical Research, Public Policy Committee: 1982 - 1986
- American Federation for Clinical Research, Publications Committee: 1983 -1986
- Clinical Immunology Society, Council Member and Executive Committee. 1992- 2011
- Clinical Immunology Society, President, 1999 -2000
- Clinical Immunology Society, Publications Committee, 1987 - 1998, Chair, 1995 - 1998
- Dartmouth Medical School Admissions Support Committee, 1983 - 1988

- National Institute of Allergy and Infectious Diseases: Co-chair for Allergy & Immediate Hypersensitivity 1996-7: Member 1989 - 1990
  Task Force on Asthma and Other Allergic Diseases/New Initiatives in Immunology
- National Institute of Allergy and Infectious Diseases: Expert Panel on the Extramural Asthma and Allergy Research Program, Co-Chair, 2000
- Pan-American Group on Immunodeficiency, Co-founder and Member of Executive Council 1995 - present
- National Institute of Allergy and Infectious Diseases: Expert Panel on Food Allergy Research, Co-Chair, 2003
- Task Force on Asthma and Other Allergic Diseases/New Initiatives in Immunology NIH, 2002
- U.S. Judiciary impartial expert witness: Swine Flu Litigation, 1984 - 1988

**Research Peer Review Funding Responsibilities (selected):**

- Immune Tolerance Network, Member and Co-Chair, Section on Allergy, Member of National Steering Committee and National Executive Committee, 2000 - 2014
- Advisory Council ad hoc member, National Institute of Allergy and Infectious Diseases, 1991
- Chair of Asthma, Allergy and Immunologic Disease Center Review, National Institute of Allergy and Infectious Diseases, 1994 and 1999.
- Co-chair of Human Immunology Think Tank, National Institute of Allergy & Infectious Diseases, 2001
- Reviewer:
  American Arthritis Foundation
  Human Frontier Science Program Organization (Europe
  Medical Research Council (Australia)
  Medical Research Council (Canada)
  Medical Research Council (United Kingdom)
  National Science Foundation
  NIH Study Sections: Ad Hoc: Immunobiology, Allergy and Immunology, Immunological Sciences, Experimental Immunology
  Veterans' Administration: Immunology Merit Review Board

**Journal Stewardship:**

- Editor-in-Chief: Clinical Immunology, 1999 – 2010,
- Special Editor/Editor emeritus, Clinical Immunology, 2011 - present
- Founder and Editor-in-Chief: AIDS Medical Update, 1983 - 1992
- Executive Editor: Clinical Immunology Spectrum, 1991 - 1996
- Associate Editor-in-Chief: The Immunologist, 1996 - 1999
- Associate Editor: AIDS Targeted Information Newsletter, 1987 - 1993
- Associate Editor: Journal of Immunology, 1982 - 1987
- Founder and Consulting Editor: AIDS Nursing Update, 1987 - 1992
- Associate Editor-in-Chief: Clinical Immunology & Immunopathology - 1998
- Editorial Board:
  Journal Allergy and Clinical Immunology, 1981 - 1986
  Journal of Clinical Immunology, 1982 - 1989
  Clinical Research, 1984 - 1986
  Disease Markers, 1985 – 2012
- Member, Committee on Publishing Ethics, Elsevier 2008-2013

**TRAINING/TEACHING ACTIVITIES (selected):**

- Co-Director: Combined Fellowship in Pediatric and Adult Immunology and Allergy, 1979 - 2003
- Director: Fellowship Program in Adult Clinical Immunology/Allergy (CIA), UCLA School of Medicine, 1977 - 1979
- American Academy of Allergy/Immunology: Trainee Award Committee Member, 1989 - 1991
- Member: Training Directors Committee, American Academy of Allergy and Immunology, 1980 - 2000
- Member: Training Directors Committee, American College of Allergy and Immunology, 1989 - 2000
- Chairman: UCLA Department of Medicine Honors Preceptor Program, 1984 - 1987
- Organizer and/or Instructor: various courses in Medicine and Microbiology & Immunology, as well as in continuing medical education, 1977 - present

**PUBLIC SERVICE:**

- Asthma and Allergy Foundation of America (Los Angeles Chapter): Board of Directors, 1981 - 1985
- Asthma and Allergy Foundation of America (Los Angeles Chapter): Medical Advisory Board, 1986 - 1999; Chairman, 1990 - 1999
- FDA Advisory Panel member - Allergenic products 1997 - 2002
- Immune Deficiency Foundation of America: Medical Advisory Board, 1990 - 2004
- PADI Foundation: Board of Directors, 1991- present, Secretary and Chief Financial Officer 1992 – present.
- Chair, USC Children's Center Advisory Committee 1999-present
- Chair, Food Allergy Initiative Medical Advisory Board 2001 – 2006
- External reviewer of the FDA research programs in the Laboratory of Immunobiochemistry, Division of Bacterial, Parasitic and Allergenic Products, Center for Biologics Evaluation and Research

**PROFESSIONAL SOCIETIES:**

- Association of American Physicians
- American Society for Clinical Investigation
- American Federation for Clinical Research
- American Association of Immunologists
- American Academy of Allergy and Clinical Immunology
- American College of Allergy and Immunology
- Clinical Immunology Society
- Collegium Internationale Allergologicum
- Los Angeles Society of Allergy and Clinical Immunology
- Pan-American Group on Immunodeficiency
- Society of Toxicology, Immunotoxicology Section
- Western Association of Physicians

**HONORS: (selected)**

- Recipient: Allergic Diseases Academic Award; National Institute of Allergy and Infectious Diseases, 1978 – 1983.
- University Visiting Professorship: Singapore Post Graduate School of Medicine and Singapore General Hospital, Singapore, 1985.
- Swineford Memorial Lectureship: University of Virginia, Charlottesville, VA, 1987.
- Catholic University Visiting Lectureship in Immunology: Catholic University, Santiago, Chile, 1987
- Rabiner Visiting Professor: University of Oregon School of Medicine, Good Samaritan Hospital, Portland, OR, 1988.
- UCLA Visiting Professorship: Harbor General Hospital/UCLA, Torrance, CA, 1988.
- Robert Orange Memorial Lectureship, University of Toronto, Hospital for Sick Children, Toronto, Canada, 1988.
- Visiting Professor, National Jewish Center for Immunology, Denver, CO, 1990.
- American College of Allergy & Immunology Visiting Professor, University of Oklahoma, Tulsa, OK, 1991.
- American College of Allergy & Immunology Visiting Professor, Temple University, Philadelphia, PA., 1992.
- Goodman Family Visiting Professor, Santa Monica Hospital Medical Center, Los Angeles, CA 1994
- UCLA Medical Sciences Research Award Recipient, 1994.
- University of Illinois College of Medicine Visiting Professor 1995.
- Keynote speaker First Japanese Clinical Immunology and Allergy Society meeting and International Guest of Honor, Japanese Society of Immunology, Allergy and Otolaryngology, 1996.
- Arbesman Commemorative Lecturer - American Academy of Allergy, Asthma and Immunology, 1997.
- National Institute of Allergy and Infectious Diseases 50$^{th}$ Anniversary Symposium Speaker, ATS, 1999.
- Recipient, Stein Oppenheimer Award 1999.
- William Pierson Lectureship, American Academy of Allergy, Asthma and Immunology, Scripps Mercy Hospital, 2000.
- First Annual Feng Pao Hsii Lectureship, Singapore Society of Immunology, Rheumatology & Allergy Society, 2000.
- Keynote Speaker, Keystone Symposium on Asthma, Sante Fe, New Mexico, Feb. 2002
- Distinguished Visiting Professor in Immunology, Biology of Inflammation Center at Baylor College of Medicine, Feb. 2004.
- Presidential Award, Clinical Immunology Society, 2005.
- Physician-of-the-Year honoree, Asthma and Allergy Foundation of America, Los Angeles Chapter, 2005.
- Carolyn and Warren Richards Allergy Lectureship, Children's Hospital of Los Angeles, Keck School of Medicine, University of Southern California, 2006.
- Distinguished Service Award, Clinical Immunology Society, 2008

## PUBLICATIONS

### Research Papers - peer reviewed

1. **Saxon, A**. and Higham, C.W.F. Identification and interpretation of growth rings in the secondary dental cementum of Ovis aris L. *Nature*. 219:634, 1968.
2. **Saxon, A**. and Higham, C.W.F. A new research method for economic prehistorians. *American Antiquity*. 34:303, 1969.
3. Ferm, V.H. and **Saxon, A**. Amniotic fluid volumes in experimentally induced renal agenesis and anencephaly. *Experimentia*. 27:1066, 1971.
4. Ferm, V.H., **Saxon, A**. and Smith, B.M. The teratogenic profile of sodium arsenate in the golden hamster. *Arch Environ Health*. 22:557, 1971.
5. Hoefnagel, D., Pomeroy, J., Wurster, D. and **Saxon, A.** Congenital athetosis, mental deficiency, dwarfism and laxity of skin and ligaments. *Helv Paediat Acta.* 26:397, 1971.
6. **Saxon, A**., Busch, G.J., Merrill, J.P. and Wilson, R. Renal transplantation in primary hyperoxaluria. *Arch of Internal Med.* 133:464, 1974.
7. **Saxon, A**. and Kattlove, H.E. Platelet inhibition by sodium nitroprusside, a smooth muscle inhibitor. *Blood*. 46:957, 1976.
8. **Saxon, A**., Feldhaus, J., and Robins, R.A. Single step separation of human T and B cells using AET treated SRBC rosettes. *J Immunol Methods.* 12:285, 1976.
9. Cline, M.J., Opelz, G., **Saxon, A**., Fahey, J.L. and Golde, D.W. Autoimmune panleukopenia. *N Engl J Med.* 295:1489, 1976.
10. O'Toole, C., **Saxon, A**. and Bohrer, R. Human lymph node lymphocytes fail to effect lysis of antibody coated target cells. *Clin Exp Immunol.* 27:165, 1977.
11. Bonavida, B., Robins, R.A. and **Saxon, A.** Lectin-dependent cellular cytotoxicity in man. *Transplantation*. 23:261, 1977.
12. Golde, D.W., **Saxon, A**. and Stevens, R.H. Macroglobulinemia and hairy-cell leukemia. *N Engl J Med*. 296:92, 1976.
13. Golde, D.W., Stevens, R.H., Quan, S.G. and **Saxon, A**. Immunoglobulin synthesis in hairy cell leukemia. *Brit Journal Haematology*. 35:359, 1977.
14. **Saxon, A**. and Portis, J. Lymphoid subpopulation changes in regional lymph nodes in squamous head and neck cancer. *Cancer Res*. 37:1154, 1977.
15. **Saxon, A**. and Portaro, J. Preservation of *in vitro* biologic functions in regional lymph node lymphocytes in squamous head and neck cancer. *Cancer Res*. 37:1159, 1977.
16. **Saxon, A**., Morledge, D. and Bonavida, G. Histamine receptor leukocytes (HRL): Organ and lymphoid subpopulation distributions in man. *Clin Exp Immunol*. 28:394, 1977.
17. **Saxon, A**., Stevens, R.H., Ashman, R.A. and Parker, N. Dual immune defects in non-granulomatous ulcerative jejunoileitis with hypogammaglobulinemia. *Clin Immunol Immunopath*. 8:272, 1977.
18. **Saxon, A**., Stevens, R.H. and Ashman, R.A. Regulation of immunoglobulin production in human peripheral blood leukocytes: Cellular interactions. *J. Immunol*. 118:1872, 1977.
19. Pratt, H.P.M., Fitzgerald, P.A. and **Saxon, A**. Synthesis of sterol and phospholipid induced by the interaction of phytohemagglutinin and other mitogens with human lymphocytes and their relation to blastogenesis and DNA synthesis. *Cell Immunol.* 32:160, 1977.
20. **Saxon, A.**, Stevens, R.H., Raimer, S.J., Clements, P.J. and Yu, D.T.Y. Glucocorticoids administered *in vivo* inhibit human suppressor T lymphocyte function and diminish B lymphocyte responsiveness in *in vitro* immunoglobulin synthesis. *J Clin Invest*. 61:922, 1978.

21. **Saxon, A**., Stevens, R.H., Quan, S.G. and Golde, D.W. Immunologic characterization of hairy cell leukemias in continuous culture. *J Immunol*. 120:777, 1978.
22. Fiala, M., Chatterjee, S.N., Carson, S., Poolsawat, S., Heiner, D.C., **Saxon, A**. and Guze, L.B. Cytomegalovirus retinitis secondary to chronic viremia in phagocytic leukocytes. *Amer J Ophthalmol*. 84:567, 1977.
23. Fiala, M. Chatterjee, S., Ellis, R., Imparato, B., Bahna, S. and **Saxon, A**. Fever of undetermined origin: Role of cytomegalovirus and Epstein-Barr virus. *West J Med*. 129:263-266, 1978.
24. Pratt, H.P.M., **Saxon, A**. and Graham, M.L. Membrane lipid changes associated with malignant transformation and normal maturation of human lymphocytes. *Leukemia Research*. 2:1, 1978.
25. **Saxon, A.** and Stevens, R.H. Suppression of immunoglobulin production in normal human blood characterization of the cells responsible and mediation by a soluble T lymphocyte derived factor. *Clin Immunol Immunopath*. 10:427, 1978.
26. Stevens, R.H., and **Saxon, A.** Anti-human helper T-lymphocyte antiserum: Generation and functional characterization. *Clin Immunol Immunopath*. 10:438, 1978.
27. **Saxon, A.**, Stevens, R.H. and Golde, D.W. T-lymphocyte variant of hairy cell leukemia. *Ann Int Med*. 88:323, 1978.
28. Stevens, R.H. and **Saxon, A**. Immunoregulation in humans: Control of anti-tetanus toxoid antibody production after booster immunization. *J Clin Invest*. 62:1154, 1978.
29. Stevens, R.H., Thiele, C. and **Saxon, A**. The production of a soluble human T lymphocyte derived factor which substitutes for helper T lymphocytes in the *in vitro* production of immunoglobulin. *Immunology*. 36:407, 1979.
30. **Saxon, A**. and Stevens, R.H. Human T lymphocyte-derived helper factor (HHF): Cellular and physical characterization. *Clin Immunol Immunopath*. 12:82, 1979.
31. Fitchen, J.H., Cline, M.J., **Saxon, A**. and Golde, D.W. Serum inhibitors of hematopoiesis in a patient with aplastic anemia and systemic lupus erythematosus: Recovery after exchange plasmaphoresis. *Amer J of Med*. 66:537, 1979.
32. **Saxon, A**., Stevens, R.H. and Golde, D.W. Helper and suppressor T-lymphocyte leukemia in Ataxia Telangiectasia. *N Engl J Med*. 300:700, 1979.
33. Stevens, R.H. and **Saxon, A**. Reduced *in vitro* production of anti-tetanus toxoid antibody after repeated *in vivo* immunization with tetanus toxoid. *J Immunol*. 122:592, 1979.
34. Stevens, R.H. and **Saxon, A**. Differential synthesis of IgM and IgG antitetanus toxoid antibody *in vitro* following *in vivo* booster immunization of humans. *Cell Immunol*. 45:142, 1979.
35. Stevens, R.H., Macy, E., Morrow, C. and **Saxon, A.** Characterization of a circulating sub-population of spontaneous anti-tetanus toxoid antibody producing B cells following *in vivo* booster immunization. *J Immunol*. 122:2498, 1979.
36. Bluming, A., Cohen, H. and **Saxon, A**. Angioimmunoblastic lymphadenopathy with dysproteinemia: A pathogenetic link between physiologic lymphoid proliferation and malignant lymphoma. *Amer J Med*. 67:421, 1979.
37. Krantman, H.J., Stiehm, E.R., Stevens, R.H., **Saxon, A.** and Seeger, R.C. Abnormal B cell differentiation and variable increased T cell suppression in immunodeficiency with hyper-IgM. *Clin Exp Immunol*. 40:147, 1980.
38. Ashman, R.F., **Saxon, A**. and Stevens, R.H. Profile of multiple lymphocyte functional defects in acquired hypogammaglobulinemia derived from *in vitro* cell recombination analysis. *J Allergy Clin Immunol*. 65:242, 1980.
39. **Saxon, A**. and Stevens, R.H. Stimulation and regulation of human IgE production *in vitro* using peripheral blood lymphocytes. *Clin Immunol Immunopath*. 14:474, 1979.

40. **Saxon, A**., Kobayashi, R.H., Stevens, R.H., Stiehm, E.R. and Siegel, S.C. *In vitro* analysis of humoral immunity with normal immunoglobulins in antibody deficiency. *Clin Immunol Immunopath.* 17:235, 1980.
41. Stevens, R.H., Tamaroff, M. and **Saxon, A.** Inability of patients with common variable hypogammaglobulinemia to generate lymphoblastoid B cells following booster immunization. *Clin Immunol Immunopath.* 16:336, 1980.
42. **Saxon, A**., Morrow, C. and Stevens, R.H. Subpopulations of circulating B cells and regulatory T cells involved in *in vitro* immunoglobulin E production in atopic patients with elevated serum immunoglobulin E. *J Clin Invest.* 65:1457, 1980.
43. **Saxon, A**., Tamaroff, M.A., Morrow, C. and Stevens, R.H. Impaired generation of spontaneous and mitogen-reactive anti-tetanus toxoid antibody producing B cells following repetitive *in vivo* booster immunization. *Cell Immunol.* 59:82, 1981.
44. Boxer, R.J., Sofen, H. and **Saxon, A**. The detection of transitional cell bladder cancer antigens on established cell lines. *Invest Urology.* 19:70, 1981.
45. **Saxon, A**., Kaplan, M. and Stevens, R.H. Isotype specific human B lymphocytes that produce immunoglobulin E *in vitro* when stimulated by pokeweed mitogen. *J Allergy Clin Immunol.* 66:231, 1980.
46. Stevens, R.H. and **Saxon, A.** Antigen-induced suppression of human *in vitro* pokeweed mitogen stimulated antibody production. *Cell Immunol.* 55:85, 1980.
47. Foon, K.A., Naiem, F., **Saxon, A**., Stevens, R.H. and Gale, R.P. Leukemia of T-helper lymphocytes: Clinical and functional features. *Leukemia Research.* 5:1, 1981.
48. **Saxon, A**., Thiele, C.J., Moroz, C. and Stevens, R.H. Adenosine receptor lymphocytes in humoral immunodeficiency. *J Clin Immunol.* 1:131, 1981.
49. Krantman, H.J., **Saxon, A.**, Stevens, R.H. and Stiehm, E.R. Phenotypic heterogeneity in X-linked infantile agammaglobulinemia with *in vitro* monocyte suppression of immunoglobulin synthesis. *Clin Immunol Immunopath.* 20:170, 1981.
50. Church, J.A., Isaccs, H., **Saxon, A**., Keens, T.G. and Richards, W. Lymphoid interstitial pneumonitis and hypogammaglobulinemia in children. *Amer Rev Resp Dis*. 124:491, 1981.
51. Cannon, R.A., Blum, P.M., Ament, M.E., Byrne, W.J., Soderberg-Warner, M., Seeger, R.C., **Saxon, A.** and Stiehm, E.R. Reversal of enterocolitis-associated combined immunodeficiency by plasma therapy. *J Pediatrics.* 101:711, 1982.
52. **Saxon, A**., McIntyre, R.E., Stevens, R.H. and Gale, R.P. Lymphocyte dysfunction in chronic graft versus host disease. *Blood*. 58:746, 1981.
53. Ahmed, R.A., Sofen, H. and **Saxon, A.** Detection of an antisquamous antibody in multiple kertoacanthoma. *Clin Immunol Immunopath*. 22:20, 1982.
54. Gottlieb, M.S., Schroff, R., Schanker, H.M., Weisman, J.D., Fan, P.T., Wolf, R.A. and **Saxon, A**. Pneumocystis carinii pneumonia and mucosal candidiasis in previously healthy homosexual men: Evidence of a new acquired cellular immunodeficiency. *N Engl J Med.* 305:1425, 1981.
55. Barbers, R.G., Shih, W.W.H. and **Saxon, A**. *In vitro* depression of human lymphocyte mitogen response (phytohemagglutinin) by asbestos fibres. *Clin Exp Immunol.* 48:602, 1982.
56. Schwartz, A.G., Targan, S.R., **Saxon, A.** and Weinstein, W.M. Sulfasalazine-induced exacerbation of ulcerative colitis. *N Engl J Med*. 306:409, 1982.
57. Ahmed, A.R., Stevens, R.H. and **Saxon, A.** Production of antibasement membrane zone antibody by peripheral blood leukocytes bullous pemphigoid patients. *J Clin Immunol*. 2:179, 1982.
58. Thompson, L.F., **Saxon, A.**, O'Connor, R.D. and Fox, R.I. Ectro-5'-nucleotidase activity in human T cell subsets: Decreased numbers of ecto-5'-nucleotidase positive cells from both OKT4+ and OKT8+ cells in patients with hypogammaglobulinemia. *J Clin Invest.* 71:892, 1983.

59. Rohr, A., Hassner, A. and **Saxon, A.** Rhinopharynogoscopy for the evaluation of allergic-immunologic disorders. *Ann Allergy*. 50:380, 1983.
60. Ahmed, R.A., Murahata, R.I., Schroff, R.W., Stevens, R.H. and **Saxon, A**. Production of pemphigus antibody *in vitro* and analysis of T cell subsets. *J Clin Immunol*. 3:241, 1983.
61. Hassner, A. and **Saxon, A**. Inhibition of ongoing myeloma IgE synthesis *in vitro* by activated human T cells. *J Immunol*. 130:1567, 1983.
62. Schanker, H.M., Young, R., Cahan, L., Schroff, R. and **Saxon, A.** Effect of midbrain stimulus induced analgesia on immune function in humans. *J Neuroimmunol*. 5:185, 1983.
63. Denis, K.A., Wall, R. and **Saxon, A.** Human-human B cell hybridomas from *in vitro* stimulated lymphocytes patients with common variable immunodeficiency. *J Immunol*. 131:2273, 1983.
64. Hassner, A. and **Saxon, A.** Isotype-specific human suppressor T cells for IgE synthesis activated by IgE-Anti-IgE immune complexes. *J Immunol.* 132:2844, 1984.
65. Kodo, H., Gale, P. and **Saxon, A**. Antibody synthesis by bone marrow cells *in vitro* following primary and booster tetanus toxoid immunization in humans. *J Clin Invest*. 73:1377, 1984.
66. Rohr, A.S., Marshall, N.A. and **Saxon, A**. Successful immunotherapy for *Triatoma protracta*-induced anaphylaxis. *J All. Clin Immunol*. 73:369, 1984.
67. **Saxon, A.**, Hassner, A., Swabb, E.A., Wheeler, B. and Adkinson, N.F. Lack of cross-reactivity between Aztreonam, a monobactam antibiotic, and Penicillin in penicillin allergic subjects. *J Inf Dis.* 149:16, 1984.
68. **Saxon, A.** and Barnett, E. Human auto-anti-idiotypes regulating T cell mediated reactivity to tetanus toxoid. *J Clin Invest.* 73:342, 1984.
69. Ochs, H.D., Allred, R.U., Ammann, A.J., Budinger, M.D., Cowan, M.T., Fischer, S.H., Rousell, R.H., **Saxon, A**., Wara, D. and Wedgewood, R. Comparison of high dose, low dose intravenous immunoglobulin therapy in patients with primary immunodeficiency diseases. *Amer J Med.* 76:78, 1984.
70. Kanowith-Klein, S. and **Saxon, A**. Fc epsilon receptors on human cell lines and peripheral blood lymphoctyes detected by binding of IgE immune complexes. *J Clin Immunol*. 5:38, 1985.
71. **Saxon, A.**, Swabb, E. and Adkinson Jr., N.F. Investigation into the immunologic cross-reactivity of Aztreonam with other beta-lactam antibodies. *Amer J Med.* 78:19, 1985.
72. Schanker, H.M., Rajfer, J. and **Saxon, A.** Recurrent respiratory disease, azoospermia and nasal polyposis: A syndrome that mimics cystic fibrosis and immotile cilia syndrome. *Arch Int Med.* 145:2201, 1985.
73. Marshall, N., Liebhaber, M., Dyer, A. and **Saxon, A**. The prevalence of allergic sensitization to Triatoma protracta (Heteroptera: Reduviidae) in a Southern California U.S.A. Community. *J Med Entomol*. 23:117, 1986.
74. Kanowith-Klein, S. and **Saxon, A**. Regulation of ongoing IgE synthesis by human T cell supernatants derived from atopic and nonatopic donors. *Int Arch Allergy Appl Immunol*. 80:33, 1986.
75. Ashida, E.R. and **Saxon, A.** Home intravenous immunoglobulin therapy by self-administration. *J Clin Immunol*. 6:306, 1986.
76. Marshall, M.S., Chapman, M.D. and **Saxon, A.** Species specific allergens from the salivary glands of Triatominae (Heteroptera: Reduviidae). *J Allergy Clin Immunol*. 78:430, 1986.
77. Chapman, M.D., Marshall, N.A. and **Saxon, A**. Identification and partial purification of species specific allergens from Triatoma protracta (Heteroptera: Reduviidae). *J Allergy Clin Immunol*. 78:436, 1986.
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80. **Saxon, A**. and Weinstein, R. Oral administration of bovine colostrum anti-cryptosporidia antibody fails to alter the course of human cryptosporidiosis. *J Parasitology* 73:413-415, 1987.
81. Sherr, E.H., Stein, L.D., Dosch, H.M. and **Saxon, A.** IgE enhancing activity directly and selectively affects activated B cells: Evidence for a human IgE differentiation factor. *J Immunol.* 138:3836, 1987.
82. Kanowith-Klein, S., **Saxon, A.**, Uittenbogaart, C.H. Constitutive production of B cell differentiation factor-like activity by human T and B cell lines. *Euro. J. Immunol.* 17:593-598, 1987.
83. Kimata, H., Shanahan, F., Brogan, M., Targan, S. and **Saxon, A**. Modulation of ongoing human immunoglobulin synthesis by natural killer cells. *Cell Immunol.* 107:74, 1987.
84. Sherr, E.H., **Saxon, A**., Wells, J.R. Functional and phenotypic characterization of human B lymphocyte subsets isolated by unit gravity sedimentation. *Int Arch Allergy Appl Immunol.* 85:154, 1988.
85. Clement, L.T., Plaeger-Marshall, S., Hass, A., **Saxon, A.** and Martin, A.M. Bare lymphocyte syndrome: Consequences of absent class II major histocompatibility antigen expression for B lymphocyte differentiation and function. *J Clin Invest.* 81:669, 1988.
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88. Kimata, H. and **Saxon, A.** A subset of natural killer cells is induced by immune complexes to display Fc receptors for IgE and IgA and demonstrate isotype regulatory function. *J Clin Invest*. 82:160, 1988.
89. Kanowith-Klein, S., Hofman, F. and **Saxon, A**. Expression of Fc e receptors and surface and cytoplasmic IgE on human fetal and adult lymphopoietic tissue. *Clin Immunol Immunopath*. 48:214, 1988.
90. Kimata, H., Sherr, E. and **Saxon, A**. Human natural killer cells produce a late acting B cell differentiation activity. *J. Clin. Immunol.* 8:381, 1988.
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# Exhibit 2

# Damp Indoor Spaces AND HEALTH

Committee on Damp Indoor Spaces and Health

Board on Health Promotion and Disease Prevention

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NOTICE: The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competences and with regard for appropriate balance.

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The serpent has been a symbol of long life, healing, and knowledge among almost all cultures and religions since the beginning of recorded history. The serpent adopted as a logotype by the Institute of Medicine is a relief carving from ancient Greece, now held by the Staatliche Museen in Berlin.

Cover: The images for the cover design were provided by Terry Brennan. The image at the center of the design is *Stachybotrys chartarum* and the border image is *Cladosporium* on paint.

*"Knowing is not enough; we must apply.*
*Willing is not enough; we must do."*
—Goethe



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# Reviewers

This report has been reviewed in draft form by persons chosen for their diverse perspectives and technical expertise, in accordance with procedures approved by the National Research Council's Report Review Committee. The purpose of this independent review is to provide candid and critical comments that will assist the institution in making its published report as sound as possible and to ensure that the report meets institutional standards of objectivity, evidence, and responsiveness to the study charge. The review comments and draft manuscript remain confidential to protect the integrity of the deliberative process. We wish to thank the following for their review of this report:

**Diane R. Gold, MD, MPH**, Harvard Medical School and Harvard School of Public Health
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Although the reviewers listed above have provided many constructive comments and suggestions, they were not asked to endorse the conclusions or recommendations, nor did they see the final draft of the report before its release. The review of this report was overseen by **Robert B. Wallace, MD**, University of Iowa, and **John C. Bailar III, MD, PhD**, University of Chicago. Appointed by the National Research Council and Institute of Medicine, they were responsible for making certain that an independent examination of this report was carried out in accordance with institutional procedures and that all review comments were carefully considered. Responsibility for the final content of this report rests entirely with the authoring committee and the institution.

# Acknowledgments

This report could not have been prepared without the guidance and expertise of numerous persons. Although it is not possible to mention by name all those who contributed to the committee's work, the committee wants to express its gratitude to a number of them for their special contributions.

Sincere thanks go to all the participants at the workshops convened on March 26, June 17, and October 8, 2002. The intent of the workshops was to gather information regarding issues related to damp indoor spaces, health effects attributed to microbial agents found indoors, and mold- and moisture-related research. The speakers, who are listed in Appendix A, gave generously of their time and expertise to help inform and guide the committee's work.

We are deeply indebted to two hard-working people—Terry Brennan and Jeroen Douwes—who served as consultants and made major contributions to the content of this report. Special thanks are also extended to Harriet Burge, chair of the committee from its inception through October 2002, for her exceptional commitment and guidance during her tenure. The committee also thanks Ulla Haverinen-Shaughnessy and Anne Hyvärinen, who permitted excerpting of text from their doctoral dissertations. Institute of Medicine staff members Michelle Catlin, Ben Hamlin, and Michael Schneider provided valuable input and help over the course of the study. The Committee on Damp Indoor Spaces and Health, of course, takes final responsibility for all content in the report.

The committee extends special thanks to the dedicated and hard-working staff at the Institute of Medicine (IOM). The expertise and leadership of Rose Marie Martinez, director of the IOM Board on Health Promotion and Disease Prevention, helped to ensure that this report met the highest standards of quality.

Finally, the committee would like to thank the chair, Noreen Clark, for her outstanding work, leadership, and dedication to this project.

# Contents

# Executive Summary

A damp spot appears in a ceiling after an intense rainstorm; a hose loosens from a washing machine, spilling gallons of water onto a basement floor; weeks after a moldy odor is detected, a plumber finds a slow leak behind a wall. There are over 119 million housing units in the United States and nearly 4.7 million commercial buildings (U.S. Census Bureau, 2003), and almost all of them experience leaks, flooding, or other forms of excessive indoor dampness at some time.

Excessive indoor dampness is not by itself a cause of ill health, but it is a determinant of the presence or source strength of several potentially problematic exposures. Damp indoor environments favor house dust mites and microbial growth, standing water supports cockroach and rodent infestations, and excessive moisture may initiate chemical emissions from building materials and furnishings.

Indoor microbial growth—especially fungal growth—has recently received a great deal of attention in the mass media. It is a prominent feature of the breakdown of dampness control; its many possible causes include a breach of the building envelope, failure of a water-use device, and excessive indoor water-vapor generation. Occupants, health professionals, and others have wondered whether indoor exposure to mold and other agents might have a role in adverse health outcomes experienced by occupants of damp buildings. Prominent among these health outcomes is acute idiopathic pulmonary hemorrhage in infants, cases of which were reported in Cleveland, Ohio in the 1990s. Residence in homes with recent water dam-

age and in homes with visible mold (including *Stachybotrys chartarum*) was among the risk factors identified in the case infants.

Against that backdrop, the Centers for Disease Control and Prevention (CDC) asked the Institute of Medicine to convene a committee of experts. CDC provided the following charge to that committee:

> The Institute of Medicine will conduct a comprehensive review of the scientific literature regarding the relationship between damp or moldy indoor environments and the manifestation of adverse health effects, particularly respiratory and allergic symptoms. The review will focus on the non-infectious health effects of fungi, including allergens, mycotoxins and other biologically active products. In addition, it will make recommendations or suggest guidelines for public health interventions and for future basic science, clinical, and public health research in these areas.

## FRAMEWORK AND ORGANIZATION

Figure ES-1 describes the path by which water or moisture sources may lead to excessive indoor dampness and to exposures that may result in adverse health outcomes. The elements of this framework are reflected in the major topics addressed in the report:

- How and where buildings become wet, the signs of dampness, how dampness is measured, the risk factors for moisture problems, and what is known about their prevalence, severity, location, and duration (Chapter 2).
- How dampness influences indoor microbial growth and chemical emissions, the various agents that may be present in damp environments, and the influence of building materials on microbial growth and emissions (Chapter 2).
- The means available for assessing exposure to microorganisms and microbial agents that occur in damp indoor environments (Chapter 3).
- The experimental data on the nonallergic biologic effects of molds and bacteria, including the bioavailability of mycotoxins and toxic effects seen in cellular (in vitro) and animal (in vivo) toxicity studies of mycotoxin and bacterial toxin exposure (Chapter 4).
- The state of the scientific literature regarding health outcomes and indoor exposure to dampness and dampness-related agents (Chapter 5).
- Dampness prevention strategies, published guidelines for the removal of fungal growth (remediation), remediation protocols, and research on the effectiveness of various cleaning strategies (Chapter 6).
- The public health implications of damp indoor environments and the elements of a public health response (Chapter 7).

The committee faced a substantial challenge in conducting its review of these topics—research on fungi and other dampness-related agents is bur-

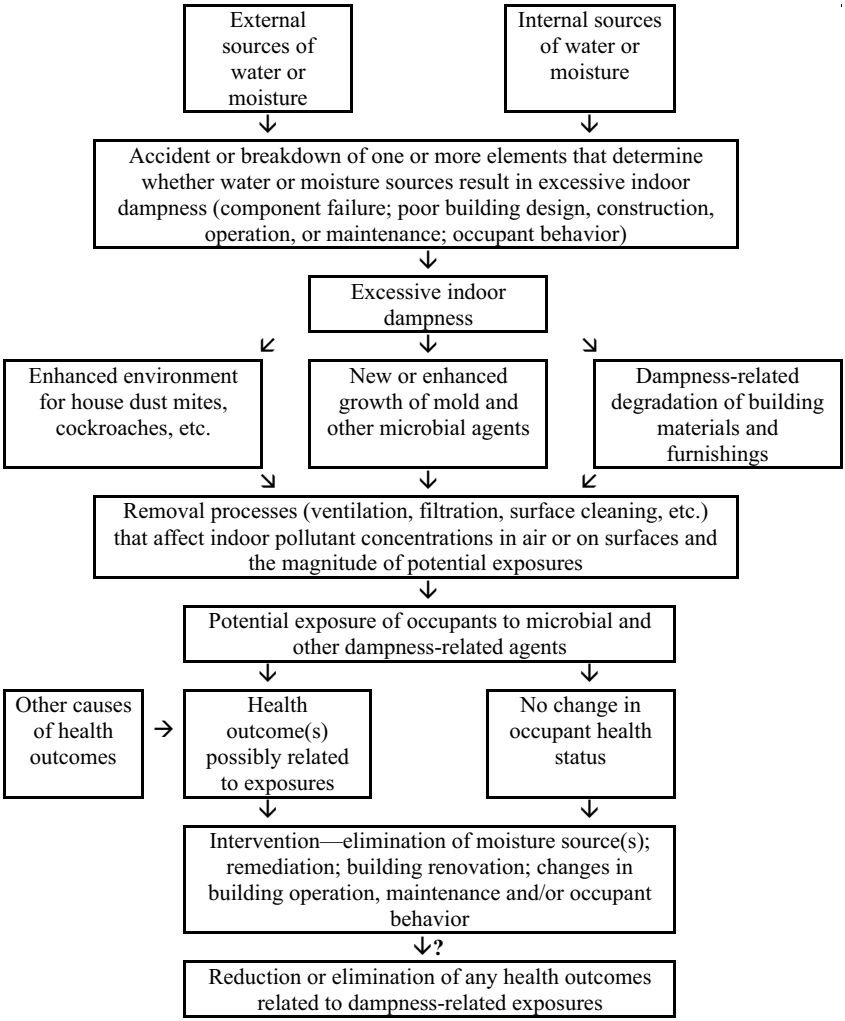


FIGURE ES-1 A framework describing the potential for water and moisture sources to lead to excessive indoor dampness and to exposures that may result in adverse health outcomes.

geoning, and important new papers are constantly being published. Although the committee did its best to paint an accurate picture of the state of the science at the time its report was completed in late 2003, it is inevitable that research advances will extend beyond the report's findings.

The sections below are a synopsis of the committee's major findings and recommendations, and the research needs they identified. Chapters 2–7 detail the reasoning underlying these and present the committee's complete findings.

## THE COMMITTEE'S EVALUATION

### Damp Buildings

The term *dampness* has been used to define a variety of moisture problems in buildings, including high relative humidity, condensation, water ponding, and other signs of excess moisture or microbial growth. While studies report that dampness is prevalent in residential housing in a wide array of climates, attempts to understand its scale and significance are hampered by the fact that there is no generally accepted definition of *dampness* or of what constitutes a "dampness problem."

There is no single cause of excessive indoor dampness, and the primary risk factors for it differ across climates, geographic area, and building types. Although the prevalence of dampness problems appears to increase as buildings age and deteriorate, the experience of building professionals suggests that some modern construction techniques and materials and the presence of air-conditioning also increase the risk of dampness problems. The prevalence and nature of dampness problems suggest that what is known about their causes and prevention is not consistently applied in building design, construction, maintenance, and use.

One consequence of indoor dampness is new or enhanced growth of fungi and other microbial agents. The fungi have (eukaryotic) cells like animals and plants, but are a separate kingdom. Most consist of masses of filaments, live off of dead or decaying organic matter, and reproduce by spores. Visible fungal colonies found indoors are commonly called mold or sometimes mildew. This report, following the convention of much of the literature on indoor environments, uses the terms *fungus* and *mold* interchangeably to refer to the microorganisms.

Mold spores are regularly found in indoor air and on surfaces and materials—no indoor space is free of them. There are a large number of species and genera, and those most typically found indoors vary by geographic area, climate, season, and other factors. The availability of moisture is the primary factor that controls mold growth indoors, since the nutrients and temperature range they need are usually present. While much attention is focused on mold growth indoors, it is not the only dampness-related microbial agent. Mold growth is usually accompanied by bacterial growth. Some research on fungi and bacteria focuses on specific compo-

nents that may be responsible for particular health effects: spores and hyphal fragments of fungi, spores and cells of bacteria, allergens of microbial origin, structural components of fungal and bacterial cells, and such products as microbial volatile organic compounds (MVOCs) and mycotoxins. Release of these components varies, depending on many physiologic and environmental factors. Dampness can also damage building materials and furnishings, causing or exacerbating the release of chemicals and nonbiologic particles from them.

Given the present state of the literature, the committee identified several kinds of research needs. Standard definitions of dampness, metrics, and associated dampness-assessment protocols need to be developed to characterize the nature, severity, and spatial extent of dampness. Precise, agreed-on definitions will allow important information to be gathered about the determinants of dampness problems in buildings and the mechanisms by which dampness and dampness-related effects and exposures affect occupant health. More than one definition may be required to meet the specific needs of health researchers (epidemiologists, physicians, and public-health practitioners) in contrast with those involved in preventing or remediating dampness (architects, engineers, builders, and those involved in building maintenance). However, definitions should be standardized to the extent possible. Any efforts to establish common definitions must be international in scope because excessive indoor dampness is a worldwide problem and research cooperation will promote the generation and dissemination of knowledge.

Research is also needed to better characterize the dampness-related emissions of fungal spores, bacteria, and other particles of biologic origin and their role in human health outcomes; the microbial ecology of buildings, that is, the link between dampness, different building materials, microbial growth, and microbial interactions; and dampness-related chemical emissions from building materials and furnishings, and their role in human health outcomes. Studies should be conducted to evaluate the effect of the duration of moisture damage of materials and its possible influence on occupant health and to evaluate the effectiveness of various changes in building designs, construction methods, operation, and maintenance in reducing dampness problems. Increased attention should be paid to heating, ventilation, and air conditioning (HVAC) systems as a potential site for the growth and dispersal of microbial contaminants that may result in adverse health effects in building occupants. And research should be performed to develop designs and construction and maintenance practices for buildings and HVAC systems that reduce moisture problems; building materials that are less prone to microbial contamination when moist; and standard, effective protocols for clean-up after flooding and other catastrophic water events that will minimize microbial growth.

## Exposure Assessment

The lack of knowledge regarding the role of microorganisms in the development and exacerbation of diseases found in occupants of damp indoor environments is due largely to the lack of valid quantitative exposure-assessment methods and knowledge of which specific microbial agents may primarily account for the presumed health effects. Very few biomarkers of exposure to or dose of biologic agents have been identified, and their validity for exposure assessment in the indoor environment is often not known. The entire process of fungal-spore aerosolization, transport, deposition, resuspension, and tracking—all of which determine inhalation exposure—is poorly understood, as is the significance of exposures to fungi through dermal contact and ingestion.

There are several methods for measuring and characterizing fungal populations, but methods for assessing human exposure to fungal agents are poorly developed and are a high-priority research need. Part of the difficulty is related to the large number of fungal species that are measurable indoors and the fact that fungal allergen content and toxic potential vary among species and among morphologic forms within species. In addition, the most common methods for fungal assessment—counting cultured colonies and identifying and counting spores—have variable and uncertain relationships to allergen, toxin, and irritant content of exposures.

Based on their review of the literature, the committee recommends that existing exposure assessment methods for fungal and other microbial agents be subjected to rigorous validation and that they be further refined to make them more suitable for large-scale epidemiologic studies. This includes standardization of protocols for sample collection, transport of samples, extraction procedures, and analytical procedures and reagents. Such work should result in concise, internationally accepted protocols that will allow measurement results to be compared both within and across studies.

The committee also identified a need to develop improved exposure assessment methods, particularly methods based on nonculture techniques and techniques for measuring constituents of microorganisms—allergens, endotoxins, $\beta(1\rightarrow3)$-glucans, fungal extracellular polysaccharides, fungal spores, and other particles and emissions of microbial origin. These needs include further improvement of light and portable personal airborne exposure measurement technology, more rapid development of measurement methods for specific microorganisms that use DNA-based and other technology, and rapid and direct-reading assays for bioaerosols for the immediate evaluation of potential health risks. Application of the improved or new methods will allow more valid exposure assessment of microorganisms and their components, which should facilitate more-informed risk assessments.

Because only sparse data are available on variation in exposure to biologic agents in the home environment, it is not possible to recommend how many samples should be taken to produce an accurate assessment of risk-relevant exposure.

### Toxic Effects of Fungi and Bacteria

Although a great deal of attention has focused on the effects of bacteria and fungi mediated by allergic responses, these microorganisms also cause nonallergic responses. Toxicologic studies, which examine such responses using animal and cellular models, cannot be used by themselves to draw conclusions about human health effects. However, animal studies are important in identifying hazardous substances, defining their target organs or systems and their routes of exposure, and elucidating their toxicokinetics and toxicodynamics, the mechanisms that account for the biologic effects, metabolism, and excretion of toxic substances. Animal studies are also useful for generating hypotheses that can be tested through studies of human health outcomes in controlled exposures, clinical studies, or epidemiologic investigations, and they are useful for risk assessment that informs regulatory and policy decisions.

Research reviewed in Chapter 4 shows that molds that can produce mycotoxins under the appropriate environmental and competitive conditions can and do grow indoors. Damp indoor spaces may also facilitate the growth of bacteria that can have toxic and inflammatory effects. Little information exists on the toxic potential of chemical releases resulting from dampness-related degradation of building materials, furniture, and the like.

In vitro and in vivo studies have demonstrated adverse effects—including immunotoxic, neurologic, respiratory, and dermal responses—after exposure to specific toxins, bacteria, molds, or their products. Such studies have established that exposure to microbial toxins can occur via inhalation and dermal exposure and through ingestion of contaminated food. Animal studies provide information on the potency of many toxins isolated from environmental samples and substrates from damp buildings, but the doses of such toxins required to cause adverse health effects in humans have not been determined. In vitro and in vivo research on *Stachybotrys chartarum* suggests that effects in humans may be biologically plausible, although this observation requires validation from more extensive research before conclusions can be drawn.

Among the other research needs identified in the chapter is further development of techniques for detecting and quantifying mycotoxins in tissues in order to inform questions of interactions and the determination of exposures resulting in adverse effects. The committee also recommends that animal studies be initiated to evaluate the effects of long-term (chronic)

exposures to mycotoxins via inhalation. Such studies should establish dose-response, lowest-observed-adverse-effect levels, and no-observed-adverse-effect levels for identified toxicologic endpoints in order to generate information for risk assessment that is not available from presently-available studies of acute, high-level exposures.

### Human Health Effects Associated with Damp Indoor Environments

The committee used a uniform set of categories to summarize its conclusions regarding the association between health outcomes and exposure to indoor dampness or the presence of mold or other agents in damp indoor environments, as listed in Box ES-1. The distinctions among categories reflect the committee's judgment of the overall strength, quality, and persuasiveness of the scientific literature evaluated. Chapter 1 details the methodologic considerations underlying the evaluation of epidemiologic evidence and details the definitions of the categories.

**BOX ES-1**
**Summary of the Categories of Evidence Used in This Report**

**Sufficient Evidence of a Causal Relationship**

Evidence is sufficient to conclude that a causal relationship exists between the agent and the outcome. That is, the evidence fulfills the criteria for "sufficient evidence of an association" and, in addition, satisfies the following criteria: strength of association, biologic gradient, consistency of association, biologic plausibility and coherence, and temporally correct association.

**Sufficient Evidence of an Association**

Evidence is sufficient to conclude that there is an association. That is, an association between the agent and the outcome has been observed in studies in which chance, bias, and confounding can be ruled out with reasonable confidence.

**Limited or Suggestive Evidence of an Association**

Evidence is suggestive of an association between the agent and the outcome but is limited because chance, bias, and confounding cannot be ruled out with confidence.

**Inadequate or Insufficient Evidence to Determine Whether an Association Exists**

The available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence of an association. Alternatively, no studies exist that examine the relationship.

Tables ES-1 and ES-2 summarize the committee's findings. The conclusions are not applicable to persons with compromised immune systems, who are at risk for fungal colonization and opportunistic infections.

Conclusions regarding exposure to agents associated with damp indoor environments are limited by the means used to assess exposure in the epidemiologic studies reviewed by the committee. For the most part, studies have relied on occupants' observations of the presence of "mold" or "moldy odor." Relatively few research efforts have used trained observers or measurements to attempt to discern which microbial agents are present, the extent of their growth, or whether there are specific common potential exposures (other than dampness). When the committee is drawing a conclusion about the association between exposure to a damp indoor environment and a health outcome, it is not imposing the assumption that the outcome is necessarily a result of exposure to a particular mold or to microbial agents in general. In some circumstances, a paper addresses the association between a particular indoor dampness-related exposure and a health outcome. However, even in those cases, it is likely that people are being exposed to multiple agents.

The committee has drawn conclusions about the state of the scientific literature regarding associations of health outcomes with two circumstances:

TABLE ES-1 Summary of Findings Regarding the Association Between Health Outcomes and Exposure to Damp Indoor Environments[a]

| | |
|---|---|
| **Sufficient Evidence of a Causal Relationship** | |
| (no outcomes met this definition) | |
| **Sufficient Evidence of an Association** | |
| Upper respiratory (nasal and throat) tract symptoms | Wheeze |
| Cough | Asthma symptoms in sensitized asthmatic persons |
| **Limited or Suggestive Evidence of an Association** | |
| Dyspnea (shortness of breath) | Asthma development |
| Lower respiratory illness in otherwise-healthy children | |
| **Inadequate or Insufficient Evidence to Determine Whether an Association Exists** | |
| Airflow obstruction (in otherwise-healthy persons) | Skin symptoms |
| Mucous membrane irritation syndrome | Gastrointestinal tract problems |
| Chronic obstructive pulmonary disease | Fatigue |
| Inhalation fevers (nonoccupational exposures) | Neuropsychiatric symptoms |
| Lower respiratory illness in otherwise-healthy adults | Cancer |
| Acute idiopathic pulmonary hemorrhage in infants | Reproductive effects |
| | Rheumatologic and other immune diseases |

[a]These conclusions are not applicable to immunocompromised persons, who are at increased risk for fungal colonization or opportunistic infections.

TABLE ES-2 Summary of Findings Regarding the Association Between Health Outcomes and the Presence of Mold or Other Agents in Damp Indoor Environments[a]

| | |
|---|---|
| **Sufficient Evidence of a Causal Relationship** | |
| (no outcomes met this definition) | |
| **Sufficient Evidence of an Association** | |
| Upper respiratory (nasal and throat) tract symptoms | Wheeze |
| Asthma symptoms in sensitized asthmatic persons | Cough |
| Hypersensitivity pneumonitis in susceptible persons[b] | |
| **Limited or Suggestive Evidence of an Association** | |
| Lower respiratory illness in otherwise-healthy children | |
| **Inadequate or Insufficient Evidence to Determine Whether an Association Exists** | |
| Dyspnea (shortness of breath) | Skin symptoms |
| Airflow obstruction (in otherwise-healthy persons) | Asthma development |
| Mucous membrane irritation syndrome | Gastrointestinal tract problems |
| Chronic obstructive pulmonary disease | Fatigue |
| Inhalation fevers (nonoccupational exposures) | Neuropsychiatric symptoms |
| Lower respiratory illness in otherwise-healthy adults | Cancer |
| Rheumatologic and other immune diseases | Reproductive effects |
| Acute idiopathic pulmonary hemorrhage in infants | |

[a]These conclusions are not applicable to immunocompromised persons, who are at increased risk for fungal colonization or opportunistic infections.
[b]For mold or bacteria in damp indoor environments.

exposure to a damp indoor environment, and the presence of mold or other agents in a damp indoor environment. As already noted, the term *dampness* has been applied to a variety of moisture problems in buildings. Most of the studies considered by the committee did not specify which agents were present in the buildings occupied by subjects, and this probably varied between and even within study populations.

The committee found **sufficient evidence of an association** between exposure to damp indoor environments and some respiratory health outcomes: upper respiratory tract (nasal and throat) symptoms, cough, wheeze, and asthma symptoms in sensitized asthmatic persons. Epidemiologic studies also indicate that there is sufficient evidence to conclude that the presence of mold (otherwise unspecified) indoors is associated with upper respiratory symptoms, cough, wheeze, asthma symptoms in sensitized asthmatic persons, and hypersensitivity pneumonitis (a relatively rare immune-mediated condition) in susceptible persons.

**Limited or suggestive evidence** was found for an association between exposure to damp indoor environments and dypsnea (the medical term for

shortness of breath), lower respiratory illness in otherwise-healthy children, and the development of asthma in susceptible persons. It is not clear whether the latter association reflects exposure to fungi or bacteria or their constituents and emissions, to other exposures related to damp indoor environments, such as dust mites and cockroaches, or to some combination thereof. The responsible factors may vary among individuals. For the presence of mold (otherwise unspecified) indoors, there is limited or suggestive evidence of an association with lower respiratory illness in otherwise-healthy children.

**Inadequate or insufficient information** was identified to determine whether damp indoor environments or the agents associated with them are related to a variety of health outcomes listed in Tables ES-1 and -2. Included among these is acute idiopathic pulmonary hemorrhage in infants (AIPHI). The committee concluded that the available case-report information constitutes inadequate or insufficient information to determine whether an association exists between AIPHI and the presence of *Stachybotrys chartarum* or exposure to damp indoor environments in general. AIPHI is a serious health outcome, and the committee encourages the CDC to pursue surveillance and additional research on the issue to resolve outstanding questions.

The committee considered whether any of the health outcomes listed above met the definitions for the categories "sufficient evidence of a causal relationship" and "limited or suggestive evidence of no association" defined in Chapter 1, and concluded that none did.

It offers some additional observations on research needs and recommendations for action:

- Indoor environments subject occupants to multiple exposures that may interact physically or chemically with one another and with the other characteristics of the environment, such as humidity, temperature, and ventilation rate. Few studies to date have considered whether there are additive or synergistic interactions among these factors. The committee encourages researchers to collect and analyze data on a broad range of exposures and factors characterizing indoor environments in order to inform these questions and possibly point the way toward more effective and efficient intervention strategies.
- The committee encourages the CDC to pursue surveillance and additional research on acute pulmonary hemorrhage or hemosiderosis in infants to resolve questions regarding this serious health outcome. Epidemiologic and case studies should take a broad-based approach to gathering and evaluating information on exposures and other factors that would help to elucidate the etiology of acute pulmonary hemorrhage or hemosiderosis in infants, including dampness and agents associated with damp indoor

environments; environmental tobacco smoke (ETS) and other potentially adverse exposures; and social and cultural circumstances, race/ethnicity, housing conditions, and other determinants of study subjects' health.

- Concentrations of organic dust consistent with the development of organic dust toxic syndrome are very unlikely to be found in homes or public buildings. However, clinicians should consider the syndrome as a possible explanation of symptoms experienced by some occupants of highly contaminated indoor environments.
- Greater research attention to the possible role of damp indoor environments and the agents associated with them in less well understood disease entities is needed to address gaps in scientific knowledge and concerns among the public.

### Prevention and Remediation of Damp Indoor Environments

Homes and other buildings should be designed, operated, and maintained to prevent water intrusion and excessive moisture accumulation when possible. When water intrusion or moisture accumulation is discovered, the source should be identified and eliminated as soon as practicable to reduce the possibility of problematic microbial growth and building-material degradation. The most effective way to manage microbial contaminants, such as mold, that are the result of damp indoor environments is to eliminate or limit the conditions that foster its establishment and growth. That also restricts the dampness-related degradation of building materials and furnishings.

Information is available on the sources of excessive indoor dampness and on the remediation of damp indoor conditions and its adverse consequences. Chapter 6 summarizes several sources of guidance on how to respond to various indoor microbial contamination situations. However, as the committee observes, determining when a remediation effort is warranted or when it is successful is necessarily subjective because there are no generally accepted health-based standards for acceptable concentrations of fungal spores, hyphae, or metabolites in the air or on surfaces.

There is a great deal of uncertainty and variability in samples of mold and other microbial materials taken from indoor air and surfaces, but the information gained from a careful and complete survey may aid in the evaluation of contamination sources and remediation needs. Visible surfaces and easily accessible spaces are not the only source of microbial contaminants, however, and the potential for exposure from sources in spaces such as attics, crawl spaces, wall cavities, and other hidden or seldom-accessed areas is poorly understood.

When microbial contamination is found, it should be eliminated by means that not only limit the possibility of recurrence but also limit expo-

sure of occupants and persons conducting the remediation. Disturbance of contaminated material during remediation activities can release microbial particles and result in contamination of clean areas and exposure of occupants and remediation workers. Containment during clean-up (through the erection of barriers, application of negative air pressure, and other means) has been shown to prevent the spread of microbial particles to noncontaminated parts of a contaminated building. The amount of containment and worker personal protection and the determination of whether occupant evacuation is appropriate depend on the magnitude of contamination.

Notwithstanding the interest in the topic, very few controlled studies have been conducted on the effectiveness of remediation actions in eliminating problematic microbial contamination in the short and long term or on the effect of remediation actions on the health of building occupants. In addition, the available literature addresses the management of microbial contamination when remediation is technically and economically feasible. There is no literature addressing situations where intervening in the moisture dynamic or cleaning or removing contaminated materials is not practicable.

Among the research needs identified by the committee are studies that better characterize the effectiveness of remediation assessment and remediation methods in different contamination circumstances, the dynamics of movements of contaminants from colonies of mold and other microorganisms in spaces such as attics, crawl spaces, exterior sheathing, and garages, and the effectiveness of various means of protection of workers and occupants during remediation activities. Standard methods should be formulated to assess the potential of new materials, designs, and construction practices to cause or exacerbate dampness problems. And research should be performed to address the other data gaps discussed above and to determine

- How free of microbial contamination a surface or building material must be to eliminate problematic exposure of occupants and in particular, how concentrations of microbial contamination left after remediation are related to those found on ordinary surfaces and materials in buildings where no problematic contamination is present.
- Whether and when microbial contamination that is not visible to the naked eye but is detectable through screening methods should be remediated.
- The best ways to open a wall or other building cavity to seek hidden contamination while controlling the release of spores, microbial fragments, and the like.
- The effectiveness of managing contamination in place by using negative air pressure, encapsulation, and other means of isolation.
- How to measure the effectiveness and health effects of a remediation effort.

## The Public Health Response

On the basis of its review of the scientific papers and other information summarized above and detailed in the report, the committee concludes that excessive indoor dampness is a public-health problem. An appropriate public health goal should thus be to prevent or reduce the incidence of potentially problematic damp indoor environments, that is, environments that may be associated with undesirable health effects, particularly in vulnerable populations. However, there are serious challenges associated with achieving that goal. As the report indicates, there is insufficient information on which to base quantitative recommendations for either the appropriate level of dampness reduction or the "safe" level of exposure to dampness-related agents. The relationship between dampness or particular dampness-related agents and health effects is sometimes unclear and in many cases indirect. Questions of exposure and dose have not, by and large, been resolved. It is also not possible to objectively rank dampness-related health problems within the larger context of threats to the public's health because there is insufficient information available to confidently quantify the overall magnitude of the risk resulting from exposures in damp indoor environments.

Institutional and social barriers may hinder the widespread adoption of technical measures and practices that could prevent or reduce problematic indoor dampness. Economic factors, for example, encourage poor practice or impede remediations; they may also create incentives to forgo or limit investment in maintenance that might help to prevent moisture problems.

Given these challenges, the committee identifies seven areas of endeavor that deserve discussion in the formulation of public health mechanisms to prevent or reduce the incidence of damp indoor environments:

- Assessment and monitoring of indoor environments at risk for problematic dampness.
- Modification of regulations, building codes, and building-related contracts to promote healthy indoor environments; and enforcement of existing rules.
- Creation of incentives to construct and maintain healthy indoor environments; and financial assistance for remediation where needed.
- Development, dissemination, and implementation of guidelines for the prevention of dampness-related problems.
- Public-health-oriented research and demonstration projects to evaluate the short-term and long-term effectiveness of intervention strategies.
- Education and training of building occupants, health professionals, and people involved in the design, construction, management, and mainte-

nance of buildings to improve efforts to avoid or reduce dampness and dampness-related health risks.

- Collaborations among stakeholders to achieve healthier indoor environments.

Among the recommendations the committee offers for implementing the actions it suggests are these:

- CDC, other public-health-related, and building-management-related funders should provide new or continuing support for research and demonstration projects that address the potential and relative benefit of various strategies for the prevention or reduction of damp indoor environments, including data acquisition through assessment and monitoring, building code modification or enhanced enforcement, contract language changes, economic and other incentives, and education and training. These projects should include assessments of the economic effects of preventing building dampness and repairing damp buildings and should evaluate the savings generated from reductions in morbidity and gains in the useful life of structures and their components associated with such interventions.
- Carefully designed and controlled longitudinal research should be undertaken to assess the effects of population-based housing interventions on dampness and to identify effective and efficient strategies. As part of such studies, attention should be paid to definitions of dampness and to measures of effect; and the extent to which interventions are associated with decreased occurrence of specific negative health conditions should be assessed when possible.
- Government agencies with housing-management responsibility should evaluate the benefit of adopting economic-incentive programs designed to reward actions that prevent or reduce building dampness. Ideally, these should be coupled with independent assessments of effectiveness.
- HUD or another appropriate government agency with responsibility for building issues should provide support for the development and dissemination of consensus guidelines on building design, construction, operation, and maintenance for prevention of dampness problems. Development of the guidelines should take place at the national level and should be under the aegis of either a government body or an independent nongovernment organization that is not affiliated with the stakeholders on the issue.
- CDC and other public-health-related funders should provide new or continuing support for research and demonstration projects that:
  - — Develop communication instruments to disseminate information derived from the scientific evidence base regarding indoor dampness, mold

and other dampness-related exposures, and health outcomes to address public concerns about the risk from dampness-related exposures, indoor conditions, and causes of ill health.

— Foster education and training for clinicians and public-health professionals on the potential health implications of damp indoor environments.

- Government and private entities with building design, construction, and management interests should provide new or continuing support for research and demonstration projects that develop education and training for building professionals (architects, home builders, facility managers and maintenance staff, code officials, and insurers) on how and why dampness problems occur and how to prevent them.
- Those formulating the education and training programs discussed above should include means of evaluating whether their programs are reaching relevant persons and, ideally, whether they materially affect the occurrence of moisture or microbial contamination in buildings or occupant health.

## REFERENCE

U.S. Census Bureau. 2003. Statistical Abstract of the United States 2002. United States Department of Commerce.

# Exhibit 3

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# REPORT OF ANDREW SAXON, M.D.

**RE: Ms. Beckemeyer vs GELCO**

## I. QUALIFICATIONS

My name is Andrew Saxon, M.D. I am a licensed physician in the State of California specializing in the area of Clinical Immunology & Allergy. I am the founder and emeritus chief of the Division of Clinical Immunology and Allergy at the UCLA School of Medicine, Los Angeles, California. I am currently a Professor of Medicine at the David Geffen School of Medicine at UCLA.

I received my medical degree from Harvard Medical School in Boston, Massachusetts, in 1972. I completed my residency in internal medicine at Harbor General Hospital in Los Angeles, California, in 1975, and my post-doctoral training in immunology in the Department of Microbiology and Immunology at the UCLA School of Medicine in 1977. I am a licensed physician in California through the National Board of Medical Examiners. I am board certified in (1) Internal Medicine by the American Board of Internal Medicine, (2) Allergy and Immunology by the American Board of Allergy and Immunology, and (3) Diagnostic Laboratory Immunology by the American Board of Diagnostic Laboratory Immunology. I founded the Division of Clinical Immunology and Allergy in the Department of Medicine at UCLA in 1977 and served as its Chairman for 30 years. I also founded the UCLA Asthma, Allergy, and Immunologic Disease Center and served as Director until the time of my retirement. I have published over 190 scientific papers in peer-reviewed journals in the field of immunology and I provide many editorial/review services for such journals including for over a decade being Editor in Chief of Clinical Immunology, the official journal of the Clinical Immunology Society. I also have served on peer-review funding committees for the National Institutes of Health and other organizations as well as chair the Allergy/Asthma section of the Immune Tolerance Network, a major undertaking by the National Institutes of Health and private institutions to discover true cures for immune mediated disorders.

I am a co-author of the American College of Occupational & Environment Medicine (ACOEM) original Position Statement entitled: Adverse Human Health Effects Associated with Molds in the Indoor Environment that appeared in 2002. ACOEM represents more than 6,000 physicians and other health care professionals and is the nation's largest medical society of individuals specializing in the field of occupational and environmental medicine. I am also a co-author of the American Academy of Allergy Asthma and Immunology's (AAAAI) official Position statement entitled "The Health Effects of Molds" which appeared in 2006. The AAAAI, with over 5000 members, is the nation's largest medical subspecialty society specifically dealing with the allergic and immune aspects of mold exposures.

Further elaboration of my professional background, prior publications and credentials is given in the attached true and correct copy of my curriculum vitae and bibliography (**Attachment "A"**).

## II. DATA RELIED UPON

A. The basis of my testimony includes my education, clinical and basic science training, experience, and review of both basic and clinical studies regarding humans and the immune system. This includes basic studies performed in the test tube and in animals regarding mold and related substances, my own research in human and animal immune reactivity, review of the exhibits, depositions and opinions of identified plaintiff experts, my extensive reading in the areas of immunology which includes allergy, autoimmunity, cancer of the immune system (lymphomas), and related areas, and my clinical experience including the diagnosis and management of patients with immunologically related disorders.

B. Included in this is extensive analysis of the literature relating to mold/fungal related illness in humans including the Institute of Medicine's 2004 Publication "Damp indoor spaces and Health" (Executive Summary provided as **Attachment "B"**), the American College of Medical Toxicology's Statement in Support of the Institute of Medicine's report (**Attachment "C"**) and the 2009 World Health Organization (2009) Guidelines for Indoor Air Quality (Executive Summary provided as **Attachment "D"**)..I am also relying upon the 2018 German review (**Attachment E**), the 2017 review by Borchers and Gershwin (**Attachment "F**"), the Position Statement of the American College of Occupational and Environmental Medicine promulgated by the Society in 2011 (**Attachment "G"**) and the 2006 Position Statement of the American Academy of Allergy, Asthma and Immunology, Asthma and Immunology (**Attachment "H"**.) I have also analyzed the 2008 General Accounting Office document entitled "*Indoor Mold: Better Coordination of Research on Health Effects and More Consistent Guidance Would Improve Federal Efforts*." (**Attachment "I"**.)

C. I am also relying upon my analysis of the case-specific materials provided to me. This includes:

C1. Medical records of the plaintiff Melanie Ann Beckemeyer PharmD from the following sources that span the timeframe March 2001 through July 2018.

- Allergy and Asthma Specialty Group.
- Bernstein Allergy Group.
- Bethesda Alcohol and Drug Treatment (no records)
- Huber personalized medicine (Dr. Gray Huber, Integrative Medicine)
- Kroger Little Clinic.
- Liberty Urgent Care.
- Dr. Harold T. Pretorius.
- Plaintiff produced 2315 pages of medical documents that include the records of Dr. Craig P. Cleveland.
- Blue Ash Family Medicine
- Report of Richard Sexton, PhD of April 2017.

C2. Environmental records

- The Report of Jeremy Porter, PMP dated 11.6.18
- Report from Ecostratum dated 7.25.18 for vehicle testing on 7.9.18

C3. Legal Documents

- Complaint filed in this matter.
- Discovery responses filed in this matter
- Depositions of Ms. Melanie Beckemeyer, PharmD. dated 9.27.18 and 11.5.18
- Expert Report of Dr. Scott McMahon in Beckemeyer vs. GELCO 8.31.18
- Expert Report of Dr. Scott McMahon in Fleming v Baker 3.29.17 (**Attachment "J"**)
- Deposition Testimony of Dr. Scott McMahon in Fleming v Baker 4.7.17 (**Attachment "K"**)
- Depositions of Scott W. McMahan in Courcelle v. CW NOLA PROPERTIES dated 8.18.17 and 5.18.18 **Attachment "L"**)
- Declaration Of Scott W. Mc Mahon, MD in Courcelle v. CW NOLA Properties Dated 8.15.17
- United States District Court Western District of Washington At Seattle, Court order 16356136 dated 8.13.15 in Haneet Kumar, et al., v. Williams Portfolio 7, Inc. (**Attachment "K")**

## III. OPINIONS

### IIIA. Opinions about Plaintiff, Ms. Melanie Ann Beckemeyer, PharmD.

Background: In this section, I will address Ms. Beckemeyer's actual medical conditions as shown by the evidence in her medical records. By way of timeframe, Ms. Beckemeyer was in possession of the vehicle in question from May 4, 2016 through September 30, 2016. She reported to her doctor that the last time she was in the car was September 23, 2016.

**Ms. Melanie Beckemeyer**, date of birth 6.5.58. Ms. Beckemeyer is 60-year old woman. She was reported to be 5'7' tall and weighed 145 lbs. on 6.26.17, the last weight I see recorded. Her medical records state that she is a non-smoker and that she would drink two to four alcoholic beverages/week. The medical records relating to Ms. Beckemeyer indicate to me that she has the following medical issues:

Hypertension- high blood pressure with secondary cognitive Issues due to small vessel ischemic changes.

Ms. Beckemeyer has had high blood pressure for many years. She was reported to have hypertension in 2001 in her first available medical record. She has been maintained on anti-

hypertensive medication (Irbesartan most recently) for a number of years and was taking that medication at the time of her 11.5.18 deposition. Common side effects of Irbesartan include dizziness, fatigue, indigestion, diarrhea and heartburn. She reported no renal disease, coronary artery disease or stroke related to her hypertension.. However, a MRI of her brain on 6.13.17 showed "*microangiopathic white matter disease consistent with chronic small vessel ischemia, most often seen in manifestation of hypertensive atherosclerosis or metabolic factors*." There was also mild right greater than left hippocampal atrophy that potentially may correlate with memory symptoms possibly related to early manifestation of mild cognitive impairment syndrome. A NeuroQuant study the same day was read as normal. Ms. Beckemeyer's records reflect she has been having concerns about her cognitive abilities since sometime in 2016. She referred herself to a neuropsychologist, Richard Sexton, PhD who saw her in May of 2017. He reported that; "*Overall, the results of the present assessment suggested that Dr. Beckemeyer is a person of above average intellectual abilities, who maintains overall cognitive/functional integrity. She demonstrated relative strengths in conceptualization, visual-spatial construction, attention, and non-verbal immediate and short-term ("working") memory/reasoning, as well as naming. She demonstrated relative weaknesses in orally-presented words/stories, delayed visual/spatial and verbal recall, and rote verbal repetition in immediate-, short-, and long-term aspects. Based on her educational and occupational/vocational history, it might be inferred that her pattern of performance might reflect a reduction from premorbid levels in the areas noted above. It is not the general pattern observed in the normal aging process or related to any emotional factor, as any reported symptoms (e.g. anxiety) were not at a disabling level. The pattern would appear to reflect the impact of* ***vascular brain changes*** *(emphasis added) or the possibility of neurotoxin exposure*."

Assessment and opinion.

It is my opinion that Ms. Beckemeyer has long-standing hypertension with underlying microvascular CNS changes resulting in a mild impairment of certain cognitive functions. Long standing hypertension with secondary microvascular changes is a very common scenario in a 63-year-old person. In contrast, exposures to molds, bacteria and their byproducts such as Ms. Beckemeyer may have had in the car in question or anywhere else are not going to involve exposure to direct neurotoxins to any significant degree. Another factor driving in Ms. Beckemeyer's cognitive complaints is her response to anxiety/stress as discussed below.

<u>Upper Respiratory and external ocular symptoms:</u> Allergic Rhinitis & Allergic Conjunctivitis,

Ms. Beckemeyer has seen an allergist, Dr. Ahmad since 2001. When he first saw her, she told him she had had "allergies" for more than 10 years. Ms. Beckemeyer had allergy testing by Dr. Ahmad in 2004. The only testing results in the records from 2004are those from intradermal testing that showed weakly positive tests to dust mites, cat, dog and "mold 2". While "prick tests are "circled" and presumably done at some point before the intradermal tests, no results of prick testing is available. Ms. Beckemeyer stated in her deposition that she received 3-4 years of allergy immunotherapy injections from Dr. Ahmad. The records of those injections and their composition is not in Dr. Ahmad's records that I received. Dr. Ahmad saw Ms. Beckemeyer on again in February 2012 and then again in 2016. He had Ms. Beckemeyer undergo allergy testing on 12.27.16. Thirty eight allergy Prick tests were done and only Epicoccum and Fusarium were recorded as "positive "at 3+ with soy at 1+ and peanut was

read as 1-2+. However, there is no reading scale for these tests nor is there a positive (histamine) or negative (saline) control result provided. Intradermal testing on that date was read as 1+ to cat and dog, 2+ to Molds 2 and 0 to Molds 1. Ms. Beckemeyer's records do not give any evidence of her having asthma. Ms. Beckemeyer has never been heard to wheeze in spite of multiple physical examinations of her lungs nor has any doctor even suggested she undergo breathing tests to look into this issue. While a number of medical records say she has "asthma", others say she does not. Ms. Beckemeyer herself testified in her deposition that she does not have asthma.

Assessment and Opinion.

It is my opinion that the data is unclear whether Ms. Beckemeyer is an allergic (atopic) person with allergic rhinitis and allergic conjunctivitis. Her history and physical examinations would indicate that is a reasonable diagnosis. However, the testing by Dr. Ahmad is problematic. The accurate form of allergy skin testing is prick testing while intradermal testing is fraught with the problem of false positive tests and particularly so in the face of multiple negative prick tests. The records I have only contain Ms. Beckemeyer's 2004 intradermal allergy skin testing, not her prick test results. However, it is reasonable to assume that the prick tests for molds, pollens, dust mites and animal dander were negative for one would never do intradermal testing to allergens that were positive on prick testing. The sets of allergy tests in December 2016 have no controls and provide no reading scales. Here the only "positive" prick tests were Epicoccum and Fusarium.

It is my opinion that, given the flaws in Dr. Ahmad's testing, I would not rely upon them to make medical diagnosis of inhalant allergy or to exclude it. I would recommend Ms. Beckemeyer gets reliable allergy blood testing done with the ImmunoCap II or equivalent third generation of allergy blood testing to determine what, if any, allergens she is allergic to.

It is my opinion that even if Ms. Beckemeyer is an allergic subject and the testing at Dr. Ahmad's office is accurate, the only allergens identified by the prick testing were Epicoccum and Fusarium and her exposure to these in the vehicle in question would have been far less than her exposure from outside air as discussed in Mr. Porter's report.

It is my opinion that Ms. Beckemeyer does not have asthma, allergic or otherwise.

<u>Status post turbonectomy and septoplasty</u>. Ms. Beckemeyer reports that she underwent a nasal turbonectomy and septoplasty prior to 2001. The reasons and date for this surgery are not elucidated in the medical records.

Assessment and opinion

It is my opinion that Ms. Beckemeyer's turbonectomy and septoplasty would have be related to cosmesis, anatomic nasal obstruction and her rhinitis (allergic or otherwise) which is discussed above..

<u>Anxiety, Panic Attacks and Depression.</u>

Ms. Beckemeyer records from Blue Ash Family Medicine document that she has had problems with depression, anxiety and panic attacks since 2011. Ms. Beckemeyer reported to those doctors that her stress in 2011 was related to both her job and her personal life. She was begun on Celexa, an antidepressant on September 8, 2011. When seen on September 26, 2011, she reported she felt that "she cannot complete tasks that require organization and

higher levels of cognition in a reasonable amount of time due to ongoing anxiety." She remained on Celexa, through 7.1.16 and was to continue it according to the records but the note from 9.7.16 does not include Celexa in the medications she is taking. The records are silent whether Beckemeyer was advised to stop Celexa by her physicians or if she stopped it on her own. Ms. Beckemeyer did not identify Celexa as a medication she was taking at the time of her deposition on 11.5.18. Ms. Beckemeyer had taken Xanax, an anxiolytic in the 1990's. She testified in her deposition that she was taking Xanax for dizziness and balance issues she was having at the time and in fact, illegally wrote herself a prescription for this drug which resulted in her being arrested and having her pharmacy license revoked for a time.

By mid 2017, Ms. Beckemeyer had developed a whole panoply of ongoing symptoms that she felt were related to her exposure to the vehicle in question which ended the previous September, The symptoms she reported on 7.5.17 included disturbed sleep, fatigue, joint pain/swelling, bladder problems, buzzing/ringing/ear pain, ,blurry vision, disorientation, forgetfulness, loss of libido, unexplained hair loss, vertigo, confusion, difficulty with concentration, and swollen glands. She endorsed mood swings/irritability/depression at that time as well.

Assessment and Opinion.

It is my opinion that Ms. Beckemeyer has a generalized anxiety disorder with panic attacks and major depressive illness. Notably, when anxious in the past, she reported cognitive symptoms very similar to what she reports now. It is my opinion that her anxiety/mood issues color and magnify her symptoms, e.g. fatigue from medications, cognitive changes etc. as well drive such symptoms on their own as such mood issues do in all patients. It is notable Ms. Beckemeyer was no longer taking her antidepressant medication in the timeframe that she began to report the increasing number of somatic symptoms, she now believes were due to exposure mold in the vehicle in question. It is my opinion that Ms. Beckemeyer likely has developed a somatic symptom disorder focused on her belief about the injury she received from her mold exposure in the vehicle in question.

<u>Vertigo/disequilibrium/dizziness:</u>

Ms. Beckemeyer reports she first had problems with vertigo and disequilibrium when she was diagnosed with Meniere's disease in her 30s. She says that she underwent endolymphatic sac surgery, reportedly in 1997, and had resolution of those symptoms. Those records are not available to me. On 9.18.15, Ms. Beckemeyer saw her primary care physician for ear pressure and being off balance that she described as feeling "swimmy." She did not report true vertigo. She also complained of nausea. At that visit, she requested a letter from the doctor to her employer as she reported that the employer arranged shuttle from the airport was causing nausea. She was treated with a short course of prednisone and a nasal steroid for presumed eustachian tube dysfunction. On 9.29.15, Ms. Beckemeyer was seen in urgent care complaining of problems with wooziness and vertigo that she attributed to have neck manipulation for Temporal Mandibular Joint (TMJ) issues. She was found to lateralize translational movements to the left on tandem positioning. Then in September of 2016, she complained of increasing light-headedness and dizziness. Over the ensuring months, she had an extensive work up for this including a neuro-otology consultation and no specific etiology was found for her ongoing complaints of light-headedness and dizziness. These symptoms persisted through the middle of 2017 at least.

Assessment and Opinion.

It is my opinion that Ms. Beckemeyer's more recent complaints of feeling dizzy and lightheaded are most likely a manifestation of both her microvascular CNS disease that is secondary to her long standing hypertension and her anxiety/stress.

Treatment with thyroid hormone Ms. Beckemeyer has been on thyroid hormone replacement medication for many years. She said in her deposition that she was started on "glandular thyroid" because someone felt she was low on thyroid. It is not clear in the records when she first started taking thyroid hormone medication nor is it clear why she was placed on it. Her anti-thyroid antibodies were negative indicating she does not have autoimmune thyroiditis, the most common cause of hypothyroidism. Ms. Beckemeyer's medical records also show that in 2017 she was taking too much thyroid hormone as documented by her very low TSH levels. Her most recent TSH level in 7.26.18 was normal.

Assessment and Opinion.

It is my opinion that if Ms. Beckemeyer actually does have underlying thyroid disease, which is not evident, it was pre-existing and any thyroid issues she may have are not related to exposure to mold, mold byproducts or ambient bacteria. It is also my opinion that overtreatment with thyroid hormone as in 2017 would exacerbate both her hypertension and her anxiety and have other potential longer-term adverse health effects.

Polycystic Ovary Syndrome with insulin resistance. Ms. Beckemeyer reported that she had been diagnosed with polycystic ovary syndrome and associated insulin resistance prior to 2012. There is no information to verify this diagnosis in the available medical records. Her records also document she has been treated with Metformin for this condition for many years. She was on metformin at the time of her deposition on 11.5.18. Her records do not give evidence of hyperglycemia or diabetes.

Assessment and Opinion. It is my opinion that should Ms. Beckemeyer have polycystic ovary syndrome; it was pre-existing and that condition is not related to exposure to mold, mold byproducts or ambient bacteria.

Glaucoma, reportedly "familial." Ms. Beckemeyer's medical records report that on 6.12.14 she reported that she had been seen for glaucoma and she stated this was "familial" There is no other information about this issue in her records.

Assessment and Opinion. It is my opinion that if Ms. Beckemeyer has glaucoma, familial or otherwise, it is unrelated to exposure to mold, mold byproducts or ambient bacteria.

Alternative health care and the pseudo-diagnosis of Chronic Inflammatory Response Syndrome, aka "CIRS." Ms. Beckemeyer has sought out a set of alternative health care providers such as Drs. Huber, Cleveland and Shoemaker/McMahon for an increasing panoply of symptoms that have persisted well after her alleged exposure ended. She has done so by self-referral as she testified to in her deposition.

Assessment and Opinion. It is my opinion that, as discussed earlier, Ms. Beckemeyer has a number of medical conditions including well-recognized psychiatric issues that contribute to her overall set of complaints. However, Ms. Beckemeyer, a PharmD. who would not be expected to be naïve in terms of what is generally accepted vs alternative/fringe medical care, has sought out medical practitioners who practice alternative/fringe medicine. Ms.

Beckemeyer self-referred herself to Dr. Huber who endorses “Shoemaker testing”, Dr. Cleveland who gave her nystatin for “mold exposure” and then sought out Dr. Shoemaker who referred her to his “trainee”, Dr. McMahon. This issue of her fringe medical care is discussed below in Section IIID below.

**IIIB. Opinions re mechanisms of adverse health effects from exposure to mold/mold products in the home and office setting as related to the plaintiff, Ms. Beckemeyer.**

Adverse health effects from mold and their byproducts occur via three main mechanisms, Immune, Infectious and Toxic/irritant (all discussed in detail in **Attachments “B” through “H”**). Each of these can be addressed in terms of whether Ms. Beckemeyer had/has evidence for an adverse health effect from mold.

i. Immune effects: There are three main possible immune mediated diseases to consider. First is typical inhalant allergy that induces allergic rhinitis and allergic asthma. Ms. Beckemeyer’s respiratory issues were discussed in detail earlier so I will not repeat that analysis here. Essentially, the medical data regarding Ms. Beckemeyer indicate she may be one of the 10% of the population who has allergic sensitization to a variety of aeroallergens including molds. However, even if she were to be, her exposure in the vehicle in question would not be the source of allergic symptoms compared to regular outdoor air. The other forms of known immune adverse health effect from exposure to mold or mold byproducts are rare conditions known as allergic bronchopulmonary aspergillosis (ABPA) and hypersensitivity pneumonitis, There is no indication of Ms. Beckemeyer having these rare illnesses.

ii. Infection: Ms. Beckemeyer has had no mold infections. In fact, the records do not even reflect that she has had problems of mold infections that are common in the general population such as mold toenail infections or skin infections. I would add that oral or vaginal candida infections are due to a yeast, Candida albicans and is not due to a mold. That yeast is resident on normal mucosal surfaces in humans but overgrows to become an infection under a variety of circumstances unrelated to ambient environmental conditions.

iii. Toxic/Irritant effects

a. Toxic effects. As discussed in **Attachments B-H, M and N,** toxic effects from inhalation of mold spores or other microbial agents in a residential setting or the vehicle in question essentially do not occur. This is simply a result of dose considerations; i) the number of spores a person can inhale, ii) the toxicity of mycotoxins on molar basis, iii) the maximum amount of mycotoxin per spore (Kelman BJ, et al. Int J Toxicol. 23:3-10, 2004, **Attachment “M“**), iv) the half-life of mycotoxins in humans, and v) the no observed effect level (NOEL) for even the most potent mycotoxins (Hardin BD, et al. J. Toxicol. Environ. Health A. 72:585-98, 2009, **Attachment “N”**). Given all these factors, it is not tenable to propose toxicity resulting from inhalation of mold spores in the air found in the vehicle in question.

b. Irritant effects. Irritant effects from mold can result from inhalation of a very large concentration of mold particulates in the air wherein the spores essentially become nuisance dust. Levels in the millions of spores/$m^3$ or more would be required for this

effect, levels far beyond anything found in the vehicle in question. Additionally, in high enough concentrations, mold derived volatile organic compounds (VOCs), e.g. gasses that smell and give rise to the musty odor associated with mold, can cause local irritant effects, usually of the wet membranes of the eye, nose and mouth. However, VOC irritant effects generally occur at levels hundreds to thousands of times higher than the odor threshold. Furthermore, even if the plaintiff had had some local irritant effects from VOCs, albeit there is no data to support that supposition, such effects would have been transient by definition, and would have disappeared within a week or two of no longer being in the vehicle in question.

Assessment and opinion.

- It is my opinion that the generally accepted mechanisms for mold and/or mold byproduct induced adverse health effects are not relevant to Ms. Beckemeyer's medical issues. , i.e. her complaints are not even potentially related to the mechanisms of accepted mold adverse health effects other than possibly some mild upper airway allergy.

**IIIB. Opinions about the medical significance of the environmental testing and potential exposures of Ms. Beckemeyer from the vehicle(s) in question.**

Background: Ms. Beckemeyer had the vehicle in question, a 2014 Toyota RAV 4 limited assigned to her from May 4, 2016 until September 30, 2016. During that time, she was living in Butler Co Ohio and working in Ohio, Kentucky, and parts of Indiana.

The relevant metric for potential human exposure to mold and mold byproducts and subsequent dose calculations is sampling of respirable air (**Attachments "B"-"H", "M" and "N".)** While testing for molds and their byproducts on surfaces in a vehicle or home or inside walls may have relevance to remediation efforts, there is no way to determine or even estimate the levels of human exposure from such sources and to attempt to do so is speculation and not generally accepted methodology for assessing human exposure. Additionally, the respirable air sampling must be in a relevant timeframe in terms of when the individuals were in the location being sampled. One cannot sample locations months or years after the individual was there and say what was present when the subject was there.

Mr. Porter's report provides an excellent summary of the Ms. Beckemeyer's history of vehicle use and an in depth analysis of the multitude of testing performed. He also discusses her residence and work locale and their potential for airborne exposures to molds and other materials. His report provides an excellent analysis of the relevance of testing for molds, bacteria and endotoxin and their potential exposures and health effects and I will not repeat that information here.

In addition to Mr. Porter's report, there is a 7.25.18 Ecostratum report regarding on testing of the vehicle in question on 7.9.18. There was only a single air sample taken. This nonviable mold spore air testing in the vehicles interior air showed 1300 spores/m3 dominated by 690 basidiospores (mushroom spores), 480 ascospores, 27 Chaetomium and 53 Cladosporium and 7 Alternaria. No Fusarium or Epicoccum spores were detected. No outdoor control sample was taken. As noted in Mr. Porter's report detailed report, these levels of are in fact low and generally less than seen many days outside in the locale where Ms. Beckemeyer lived and is living.

Assessment and Opinion

It is my opinion that the only samples that are possibly relevant to potential adverse health effects for Ms. Beckemeyer are the respirable air samples from the vehicle in question. Those samples do not give evidence that the air in the vehicle would have represented any form of health hazard to Ms. Beckemeyer.

It is my opinion that since the testing of vehicle was more than a year after Ms. Beckemeyer was in the vehicle, it is speculation to say what the levels were at the time she was using it.

It is my opinion that respirable air levels of mold spores below 6500 spores/m3 are considered "low" by the National Allergy Board and that is in terms of potential health effects for subjects allergic to the mold in question. Given that the airborne levels measured of mold spores in the vehicle in question for molds Ms. Beckemeyer may be allergic to, were in fact low and far below those found in normal outdoor air in many places throughout the USA, the air in her vehicle would not have represented any form of increased health hazard risk to her.

## IIID. Alternative/fringe health care and pseudo-diagnosis of Chronic Inflammatory Response Syndrome, aka "CIRS; Dr. Scott McMahon and Dr. Richie Shoemaker:

Dr. Scott McMahon is a pediatrician who practices medicine in Roswell, New Mexico. He practiced pediatrics until 2009 when he met Dr. Richie Shoemaker and began to practice under the title of the Whole World Healthcare, PC. Dr. McMahon states in his resume that he is "*specializing in the diagnosis and treatment of Chronic Immune Response Syndrome*." Dr. McMahon advertises himself as "Certified in Shoemaker Protocol for the Treatment of Chronic Immune Response Syndrome". Dr. McMahon appears to be proud of the fact he was the "*First (*Shoemaker*) certified practitioner in the world*" (McMahon Resume, page 1, March 2015), a very dubious distinction indeed given that Dr. Shoemaker and his diagnosis and management of his so-called *Chronic Immune Response Syndrome* are outside the widest bounds of generally accepted medicine as will be discussed below. Dr. McMahon has had no post-graduate formal education or training in immunology, clinical immunology, internal medicine, or pulmonary medicine.

Dr. McMahon wrote a report dated 8.31.18 regarding Ms. Beckemeyer's medical issues. Ms. Beckemeyer testified that he examined her in a hotel room in Indianapolis Indiana on 7.30.18. Dr. McMahon's report says he also spoke with her on the phone on 7.24.18, and again on 8.19.18.

**D1. McMahon report is** focused on his opinion that Ms. Beckemeyer has chronic inflammatory response syndrome ("CIRS") and that CIRS is the only known illness that could cause all the symptoms that Ms. Beckemeyer reports. Dr. McMahon clearly believes that the overarching diagnosis for all Ms. Beckemeyer woes is CIRS. CIRS was invented by a former practicing family physician, Dr. Richie Shoemaker. Indeed, Ms. Beckemeyer has discussed her case with Dr. Shoemaker even though he is no longer licensed to practice medicine. The report from Dr. McMahon about Ms. Beckemeyer is simply a recitation of the ideas put forth by Dr. Shoemaker who has been marketing himself and his "patent pending" diagnosis of CIRS (United States Patent Application Publication, Pub. No.: US 2014/0046143 A1 Shoemaker et al., Pub. Date: Feb. 13, 2014, Methods For Diagnosing, Treating, And Monitoring Chronic Inflammatory Response Syndrome).

As proposed by Dr. Shoemaker and regurgitated by Dr. McMahon, CIRS represents an sustained uncontrolled activation of the "innate immune system" resulting from indoor and remarkably only indoor exposure to any/all of a host of potential microbes/microbial products that results in a panoply of systemic and organ specific symptoms and signs. Additionally, they proposes that exposure to such microbes/microbial products in the future will lead to enhanced exacerbations of the subjects' CIRS. According to Drs. Shoemaker and McMahon, there is no detectable lower limit for an exposure that can induce CIRS.

Dr. Richie Shoemaker is a former family practice physician from Maryland who is a self-styled "mold expert". He withdrew from the practice medicine in 2013 while under investigation by the Maryland Medical Board. Subsequently, the Maryland Medical Board issued a highly critical reprimand letter to Dr. Shoemaker. (**Attachment "O").** The Medical Board's decision was "*should the physician resume the practice of medicine the physician will be placed on probation for minimum of two years with terms and conditions. The board found the physician failed to meet the standard of care.*" https://www.mbp.state.md.us/bpqapp/PProfile.asp. Subsequently, Dr. Shoemaker then began to market a program where for $3000; other physicians could be "certified" by Dr. Shoemaker in his protocol for diagnosis and treatment of "CIRS." Thus, Dr. Shoemaker trained and "certified" Dr. McMahon.

**D1a. Assessment and Opinions re CIRS**:

- It is my opinion that Dr. Shoemaker invented the "illness" he and others like Dr. McMahon currently call Chronic Immune Response Syndrome or "CIRS." This purported syndrome is not generally accepted entity in the medical community. CIRS is a diagnosis that is unique to Dr. Shoemaker and his followers such as Dr. McMahon. Dr. Shoemaker has applied various terms (e.g. Biotoxin illness, Building Related Illness, Wet Building Syndrome) and variable criteria to this purported illness over the past 10+ years.
- It is my opinion that Dr. Shoemaker and Dr. McMahon's allegation that CIRS is self-sustaining reaction of the innate immune system is in direct conflict with what is known about the human innate immune response.
- It is my opinion that many of the battery of tests Drs. McMahon and Shoemaker use to support their diagnosis of CIRS are not generally accepted methodology for any purpose in clinical medicine much less for the purpose for which he employs them.
- It is my opinion that Drs. McMahon and Shoemaker fail to employ the proper tests to define if the type of condition he alleges CIRS represents, e.g. a systemic inflammatory response is actually present.
- It is my opinion that the proposed treatment of CIRS with cholestyramine, Welchol, erythropoietin and pioglitazone is both inappropriate and, in the case of the latter two medications, dangerous. I will use this use of oral cholestyramine by way of example to demonstrate the lack of logical thought by CIRS practitioners. Oral cholestyramine can bind to lipophilic (lipid-liking) negatively charged substances such as cholesterol in the GI tract. Thus, it can be used to remove orally ingested negatively charged lipophilic toxins and/or toxins that recirculate via the liver and biliary system. However, the mold toxins CIRS advocates allege they are dealing with are not ingested but inhaled thus they are not in the GI tract nor are mycotoxins excreted/recirculate via the liver/biliary system. Furthermore, mycotoxins are not negatively charged molecules. Thus giving oral cholestyramine for alleged mycotoxin inhalation exposure is completely illogical.

Furthermore, the time mycotoxins remain in the body is generally less than a week so the later or long term use of oral cholestyramine, even if it was capable of removing mycotoxins (which it is not), as suggested by Drs. Shoemaker and adopted by Dr. McMahon is illogical.

- It is my opinion that Dr. Shoemaker and Dr. McMahon's proposal that immune responses in CIRS and in immunology in general fail to follow dose response relationships is profoundly mistaken and in direct contrast what is known about the immune system. All medicine including toxicology and immunology involve a dose response. For example, Dr. McMahon has alleged there is no dose response in anaphylaxis but that is completely mistaken. All of these events have a dose response even when there is an immune cascade that follows the triggering event. Immunologists clearly recognize the amplification fact of immune cascades but they are all dose dependent. Nothing Dr. McMahon, Dr. Shoemaker or others can say will alter the truth of the dose response in all of medical science as was first espoused by Paracelsus in the 15th Century.
- It is my opinion that the literature that Dr. Shoemaker and Dr. Mahon cite as actually supporting their opinions regarding CIRS has been generated by Dr. Shoemaker and his colleagues and true experts in the relevant fields do not generally accept that work as reliable.
- It is my opinion that given the shortcomings of Dr. Shoemaker's methodology, theories and diagnoses, it is not surprising that Dr. Shoemaker testimony has been excluded by the courts on at least 11 occasions as listed in **Attachment "P**"
- It is my opinion that Dr. Shoemakers whole approach was soundly criticized by the Federal Bench in the Young matter (**Attachment "Q"),** a matter in which his testimony was excluded by the Court.
- It is my overarching opinion that Dr. McMahon, in diagnosing Ms. Beckemeyer with CIRS, is not practicing generally accepted medicine.

Dr. McMahon alleges that the 2008 General Accounting Office document entitled "*Indoor Mold: Better Coordination of Research on Health Effects and More Consistent Guidance Would Improve Federal Efforts*." validates a set of diagnostic criteria for CIRS.

- It is my opinion that Dr. McMahon's statements that the GAO document validates a set of diagnostic criteria for CIRS is false. Nowhere in the entire GAO document does it mention CIRS or Dr. Shoemaker (or Dr. McMahon) and the document does not provide any form of validation of CIRS criteria. The document prepared by General Accounting Office of the Federal Government is not a scientific analysis of data. Its purpose was to analyze the research situation at a Federal level of effort. The GAO reviewed 20 written "reviews" and some government-generated documents. The conclusion of the document makes this agenda clear: "*Better coordination of research on health efforts and more consistent guidance would improve federal efforts*." Clearly, the GAO document is not a scientific contribution but an analysis of governmental undertaking in the area of indoor mold.

Dr. McMahon says that each of the lab tests he uses is approved by CLIA.

- It is my opinion that the statement by Dr. McMahon that each of the lab tests he uses is approved by CLIA is false. CLIA (the federal Clinical Laboratory Improvement Act)

simply is not involved at all in the "approval" of laboratory tests. Any expert in laboratory medicine also knows that even the FDA does not approve laboratory tests. The FDA clears test kits that are sold for diagnostic use in more than one state. In contrast, most of the tests used by Dr. McMahon to make his diagnosis of CIRS are not FDA cleared but are "home brew" tests developed by and using in an individual laboratory and thus are not subject to oversight by any governmental agency.

Dr. McMahon relies on the Dr. Shoemaker's interpretation of HLA-Dr (a set immune response gene loci) of Ms. Beckemeyer to categorize her as susceptible to and having CIRS.

- It is my opinion that Dr. Shoemaker and McMahon's purported HLA-Dr typing categorization as to susceptibility to mold related or other processes is completely baseless and has no dipositive value.

Dr. McMahon, in his report, says that Ms. Beckemeyer's NeuroQuant® test of 6.13.17 shows an "increase in the forebrain parenchyma size and atrophy of the caudate nucleus and pallidum. Her mold scoring would be 4 of a possible 8 which is a positive result for structural brain damage caused by CIRS".

Assessment and Opinion:

- It is my opinion that the NeuroQuant® test is not generally accepted as a valid measure of mold exposure or mold related CNS effects. The (mis)use of this test in making the pseudo-diagnosis of CIRS is another misadventure popularized by Dr. Richie Shoemaker much like his misuse of HLA-Dr typing. Parenthetically, the official results of Ms. Beckemeyer 6.13.17 NeuroQuant® test for accepted measurements show readings within the age-matched reference charts, e.g. a normal result.
- Notably, neither the Institute of Medicine nor the World Health Organization found evidence linking exposure to mold or dampness with any neurological adverse end points. (**Attachments "B" and "D"**). Dr. Paul Lees-Haley nicely summarized the issue of Mold Neurotoxicity in an article entitled *Mold Neurotoxicity: Validity, Reliability and Baloney* http://users.physics.harvard.edu/~wilson/soundscience/mold/lees.html.

Drs. McMahan alleges that Ms. Beckemeyer's serum MSH level is low at 5 pg/mL Dr. McMahon alleges that her "low" MSH is part of CIRS. Dr. McMahan and Dr. Shoemaker claim that lower limit of the reference value for MSH is 35 pg/mL even though the Laboratory doing the tested reports a reference range of 0 (i.e., below the limit of detection) to 100 pg/mL.

Assessment and Opinion:

- It is my opinion Ms. Beckemeyer's serum MSH level is well within the true reference range as defined by the laboratory doing the test.
- It is my opinion that Drs. McMahan and Shoemaker have arbitrarily defined their own "normal" values for serum MSH, and in doing so, ignore the reference values as defined by the laboratory doing the test. What Drs. McMahon and Shoemaker have done is far outside the bounds of generally accepted practice. While they allege that the laboratory reference range of 0 (i.e., below the limit of detection) to 100 pg/mL

provided by the testing laboratory is wrong, they provide no real data to support their belief that the lower limit of the reference value for MSH should be 35 pg/mL.

Dr. McMahon says that there no other single disease other than CIRS that could be responsible for all of Ms. Beckemeyer's myriad of complaints.

- It is my opinion that there is no single true pathophysiologic disease that encompasses all of Ms. Beckemeyer's medical issues; Indeed, Ms. Beckemeyer has a number of distinct medical issues, each of which should be addressed individually. Such a set of different disease processes is typical for 60-year-old individuals.
- It is my opinion that Dr. McMahon's belief that CIRS can explain everything that troubles Ms. Beckemeyer (or anyone else) is based on the fact the hypothetical construct of CIRS has no boundaries. They use the rubric of CIRS such that it can be applied to any subject to explain any set of issues, i.e. is all-inclusive and thus becomes meaningless as a specific disease process.

## IIIE. Summary of Opinions.

- **It is my overarching opinion that Ms. Beckemeyer's medical complaints and issues past or present were or are not due to exposure to molds, bacteria or mold/bacteria byproducts that she may have experienced related to the vehicle in question..**
- **It is my overarching opinion that the environmental testing of respirable air in Ms. Beckemeyer's vehicle does not give evidence that the level of mold spores indicate any form of increased health hazard to Ms. Beckemeyer.**
- **It is my overarching opinion that Dr. Scott McMahon does not practice generally accepted medicine in making the pseudo-diagnosis of CIRS. In doing so, he follows the fatally flawed practices of Dr. Richie Shoemaker, a former family practice physician from Maryland who is a self-styled mold "expert" and who withdrew from the practice medicine when under investigation and about to be sanctioned by the Maryland Medical Board.**
- **The opinions expressed in this report are more probable than not.**
- **The opinions expressed in the report are based on the data available to me at the time of this writing. Should new data become available, I reserve the right to modify the opinions in this report based on that new information.**

## IV. PUBLICATIONS:

All of my publications are listed in my CV/bibliography (**Attachment "A"**), are available in the public domain, and can be accessed via PubMed at https://www.ncbi.nlm.nih.gov/pubmed/ .

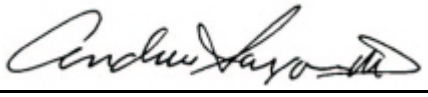
## V. PRIOR TESTIMONY

A list of my testimony in the past four years is provided as **Attachment "R"**.

## VI. FEES

My compensation for preparation of this report is at a rate of $720/hour. My fees are $720/hour for consultation time and $900/hour for testimony time.

Respectfully submitted,



Andrew Saxon, MD

November 22, 2018

Date